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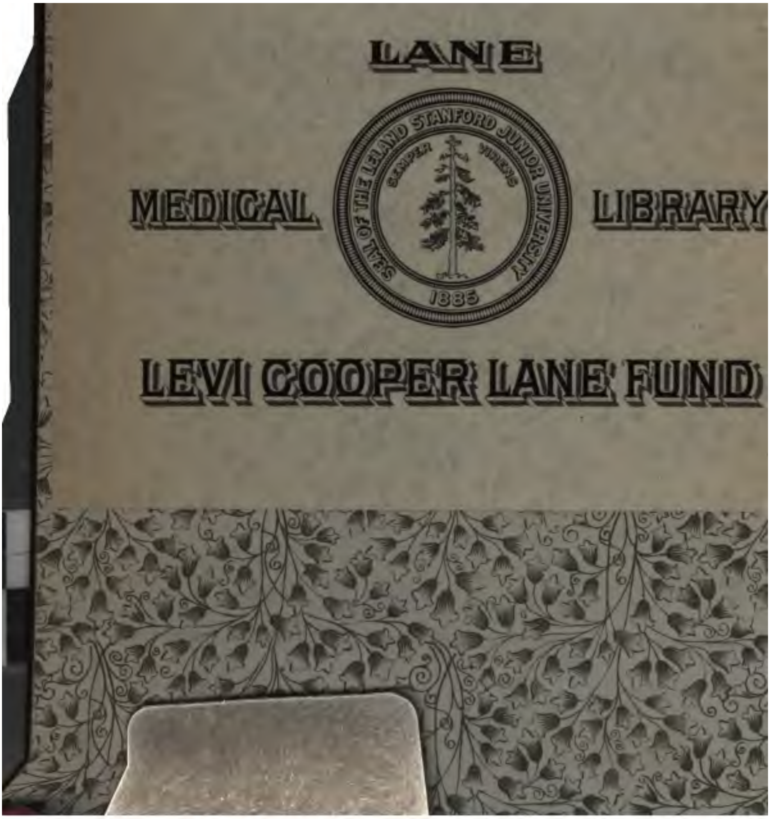

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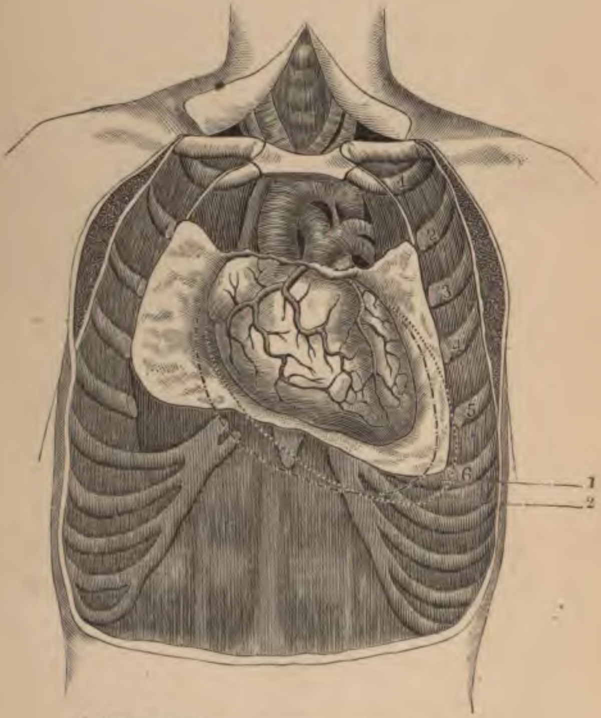

1 Area of hypertrophy of right or left ventricles.
2 Area of dilatation.

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FOR
THE STUDENT AND PHYSICIAN.

BY

DR. EDWARD T. BRUEN,

ONE OF THE PHYSICIANS TO THE PHILADELPHIA HOSPITAL, AND DISPENSARY OF THE CHILDREN'S HOSPITAL; DEMONSTRATOR OF CLINICAL MEDICINE AND LECTURER ON PATHOLOGY OF THE URINE, IN THE UNIVERSITY OF PENNSYLVANIA; LECTURER ON PATHOLOGY IN THE WOMAN'S MEDICAL COLLEGE OF PHILADELPHIA, ETC.

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PREFACE.

When the processes of morbid physiological action have produced structural changes in the tissues forming the various organs and framework of the body, the science of physics, which takes cognizance of the changeful properties of matter, enables us to institute methods of examination to which the term physical diagnosis forms a clear and appropriate designation. The purely objective physical signs are so closely intertwined with the general symptoms of disease, that any study of diagnosis is partial which does not recognize this fact. Moreover, both physical signs and general symptoms must be in their turn considered in connection with a thoughtful analysis of the processes of morbid anatomy, together with the broad principles of pathological research.

It cannot be too often repeated that symptomatology is the study of the expression of pathological changes. This conception lies at the foundation of the principles and practice of diagnosis. It is to be remembered that no single fact determined by the methods of physical diagnosis has special pathological significance, but simply indicates certain definite physical conditions of the organ under examination. For some years the writer has been engaged in teaching diagnosis to private classes of post-graduates and others; this handbook merely contains the substance of the instruction given.

It is offered to students of bedside diagnosis and clinical medicine, in the hope that it may be of assistance in welding together information gleaned from didactic lectures and reading, on the one hand, and that obtained from bedside study on the other hand.

The subject is treated in as simple and practical a manner as possible, without discussion of questions of historical or theoretical interest, and without laying special claim to originality of matter.

The author would like to express his thanks to his friend, Dr. John M. Keating, of Philadelphia, to whom he is indebted for the execution of the drawings which have been used in illustration of the text.

1581 *Chestnut St.*, *Philadelphia*,
October 1*st*, 1881.

TABLE OF CONTENTS.

PART I.

CHAPTER I.

METHODS OF DIAGNOSIS, ... 17

CHAPTER II.

METHODS OF DIAGNOSIS—AUSCULTATION, . . . 27

CHAPTER III.

PRINCIPLES OF CLASSIFICATION—ACUTE, CROUPOUS AND CATARRHAL PNEUMONIAS, ... 41

CHAPTER IV.

SUBACUTE AND CHRONIC CONSOLIDATIONS—SYPHILIS IN THE LUNGS—INCIPIENT PHTHISIS—ADVANCING CONSOLIDATION, 52

CHAPTER V.

AUSCULTATION—ADVANCING CONSOLIDATION, . . 66

CHAPTER VI.

INDEPENDENT MODIFICATIONS OF RESPIRATORY RHYTHM, 76

CHAPTER VII.

ADVENTITIOUS CAVITIES IN THE PULMONARY SUBSTANCE, 79

CHAPTER VIII.

EMPHYSEMA, ... 89

CHAPTER IX.

RALES—BRONCHITIS, ACUTE AND CHRONIC.—ASTHMA—RENAL ASTHMA—ACUTE MILIARY TUBERCULOSIS, . . 99

CHAPTER X.

DIAGNOSIS OF THE LESIONS AFFECTING THE PLEURAL CAVITIES—ACUTE PLEURISY—INCREASING EFFUSION—HYDROTHORAX, ... 123

CHAPTER XI.

CHRONIC PLEURISY, ... 140

CHAPTER XII.

EMPYEMA—PNEUMOTHORAX—HÆMOTHORAX, . . 144

CHAPTER XIII.

MALIGNANT DISEASES OF THE MEDIASTINUM, . . 152

CHAPTER XIV.

RESPIRATORY PERCUSSION, ... 156

PART II.

DISEASES OF THE HEART AND PERICARDIUM.

CHAPTER I.
PROCESSES WHICH DEVELOP CARDIAC DISEASES, . . 159

CHAPTER II.
DIAGNOSIS OF VALVULAR DISEASE—ASSOCIATE SYMPTOMS AND CHANGES IN THE CARDIAC SUBSTANCE, INCIDENT TO VALVULAR DISEASE—"MITRAL AND TRICUSPID LESIONS," 163

CHAPTER III.
MITRAL OBSTRUCTION, ... 177

CHAPTER IV.
AORTIC DISEASE—OBSTRUCTION—REGURGITATION—VALUE OF THRILL IN DIAGNOSIS OF VALVULAR DISEASES, . 184

CHAPTER V.
ANEURISMAL DISEASES OF THE ARTERIAL SYSTEM, . 192

CHAPTER VI.
INORGANIC OR FUNCTIONAL MURMURS IN THE HEART; THE VENOUS, THE ARTERIAL SYSTEMS, ... 203

CHAPTER VII.
FUNCTIONAL DISEASE—VARIATIONS OF RHYTHM—NEUROSAL DISEASE, ... 207

CHAPTER VIII.
THE PULSE—THE SPHYGMOGRAPH, ... 216

CHAPTER IX.
CARDIAC THROMBOSIS, ... 225

CHAPTER X.
DISEASES OF THE CARDIAC SUBSTANCE—HYPERTROPHY—DILATATION, ... 229

CHAPTER XI.
DEGENERATIONS OF THE HEART SUBSTANCE, ... 237

CHAPTER XII.
PROCESSES AFFECTING THE PERICARDIUM, . . . 244

INTRODUCTION.

The general appearances of the healthy adult chest are sufficiently familiar to need little description. In the male the shoulders should be moderately broad, square and level. In shape it is compressed antero-posteriorly, the back being comparatively flat, while anteriorly the walls of the chest present an ample lateral curve, and slope gradually upward to the clavicles. The nipples are situated in the fourth interspace, but their position is variable. In the female the shoulders are more rounded and sloping, the sides of the chest taper downward toward the waist, while in front the large accumulation of adipose tissue in the sub-clavicular regions and the prominent mammary glands conceal the form of the bony thorax.

When the soft parts are removed the bony case appears in both sexes as a truncated conoidal form, flattened antero-posteriorly with a broad base, and a more round apex, the lower end of the sternum being twice as far away from the spinal as the upper end. The greatest circumference is just below the middle. The female chest is less deep than the male. The transverse section is elliptical. The trachea enters the upper aperture of the chest in the median line, and descends opposite the third dorsal vertebra, where it divides into two bronchi, one for each lung. The right bronchus is wider, shorter and more horizontal in direction than the left, is about one inch in length, and enters the right lung opposite the fourth dorsal vertebra. The left bronchus is nearly two inches long and passes more obliquely downward, entering the root of the left lung, near the fifth dorsal vertebra. Each lung is attached a little above the middle of their inner surface to the trachea and heart by their roots, which contain in addition to the bronchus a branch of the pulmonary artery, pulmonary veins, bronchial arteries and veins, the pulmonary plexus of nerves, lymphatics, and bronchial glands. The apex of the lung forms a tapering cone extending from one to one and a half inches above the level of the first rib, slightly higher on the right side. The base broad, concave, and is opposed to the convex surface of the diaphragm; its circumference is thin and extends anteriorly down to the lower border of the sixth rib, laterally to the seventh rib, on the right side, and a little lower on the left side, and posteriorly down to the eleventh rib. The posterior border of each lung lies in the deep groove behind the spinal column, it is thick, rounded, and covers the root of the lung. The anterior borders are thin and sharp; they extend forward into the mediastinal space, so as to come into contact with each other, as far down as the fourth rib, below that level the border of the right lung in the median line of the sternum continues to encroach upon the sternal region, while on the left side it retreats from the sternum, leaving a considerable portion of the pericardium uncovered, in an irregularly sloping direction.

The pericardium is conical in shape, its base is attached to the central tendon of the diaphragm, extending a little further to the left than to the right side; its apex is directed upward, and surrounds the great vessels just beyond their origin from the base of the heart. It is situated behind the sternum and the cartilages of the third, fourth, fifth, sixth, and seventh ribs, on the left side, but it is in part covered by the anterior margins of the lungs. Its upper limit is about the lower edge of the second rib. The portion of the sac which is uncovered by the lungs is widely triangular in shape, and extends from the fourth interspace down to the sixth rib, and from the median line of the sternum out to within one-half inch of the line of the left nipple.

The heart lies obliquely in this sac, held by its attachment to the great vessels which spring from its base. This broad detached end is directed upward and backward to the right, and corresponds to the interval between the fifth and eighth dorsal vertebræ. On the anterior surface of the chest its upper border corresponds with a line drawn on a level with the upper border of the third costal cartilage. The lower border of the heart corresponds to a line drawn from the xiphoid cartilage (right costo-xiphoid articulation) along the upper border of sixth rib to the position of the apex, which is situated in the fifth interspace, an inch inside the line of the left nipple, or two and a half inches from the median line of the sternum. The heart is placed beneath the lower two-thirds of the sternum, and projects a little beyond the right sternal border.

In percussion of the præcordia we have two rudely triangular areas of resonance: one of impaired resonance, one of flatness. The area of flatness is that district over which the edges of the lungs do not meet. This area is bounded at the mid-sternal line by level of the fourth left costo-sternal articulation. Prolong a line from this point to the apex of the heart. Complete the right-angled triangle by projecting a line from the apex beat to the mid-sternal line once more. The other boundaries correspond to the area of impaired resonance. The two triangles vary slightly with the position of the patient, either sitting or standing. Percuss with the fingers held vertically, commencing a little to the right of the sternum, until dullness is recognized. Continue the percussion until beyond the apex pulmonary resonance is again reached. Then place the fingers parallel with the ribs to the left of the apex, and percuss gradually inward, interspace by interspace, till the sound changes from resonance to impaired resonance, to dullness. Afterward, by placing the finger used as a pleximeter obliquely pointing towards the sternum. Commencing at the apex, the previous percussion can be checked. The upper limit of dullness can be defined by percussion from above downward, the fingers parallel to the ribs.

The lower limit is not so easily defined, since the cardiac dullness blends with that of the left lobe of the liver, and the dullness of the two organs can scarcely be discriminated. A line of dullness can, however, be defined between the apex on the left and the commencement of liver dullness on the right.

The position of the areas of cardiac dullness is much influenced by the degree of distention of the intestinal tube. If the diaphragm is elevated, the heart is carried upward.

The *aorta* and *pulmonary artery* are the only vessels connected with the base of the heart which furnish physical signs of much importance. The aorta commences opposite the middle of the sternum, on a level with its junction to the third costal cartilage; it passes upward to the right for a distance of about two inches, almost in the direction of the heart's axis, as high as the upper border of the second right costal cartilage, and about a quarter of an inch behind the posterior surface of the sternum. It then passes from right to left, and from before backward to the left side of the second dorsal vertebra behind. In this part of its course its upper border is usually about *one inch* below the *upper margin* of the sternum. After reaching the third dorsal vertebra the aorta descends in a straight course, at first lying on the left side of the spine, but approaching the median line as it descends, until it passes through the aortic opening in the diaphragm.

The *pulmonary artery* is a short, wide vessel, about two inches long, arising from the left side of the base of the right ventricle; it crosses in front of the ascending aorta at the level of the third costal cartilage, and about at the left margin of the sternum. It extends upward, backward and slightly to the left as far as the under surface of the arch of the aorta, when it divides into the right and left pulmonary arteries. The right branch is somewhat larger than the left; it runs horizontally outward, behind the ascending aorta and superior vena cava, to the root of the right lung. The left branch passes horizontally in front of the descending aorta, and left bronchus to the root of the left lung.

It is important, also, to be familiar with the relative position of the viscera which occupy the upper portion of the abdomen, since by their encroachments upon the area of the thorax they materially modify the results of physical examination.

The liver lies transversely, its right lobe occupying the right hypochondriac region, while the left lobe reaches across about two inches (in the writer's experience, the slightly varying size of the organ, and the possibility of change of position, owing to tympanitic distention of the abdominal cavity, render various limits mentioned a possibility) beyond the median line. Its upper border is at the level of the fifth intercostal space; but, owing to the convexity of the upper surface, is separated some distance from the anterior thoracic wall. It is capped, as it were, by the concave base of the lower right pulmonary lobe. It comes in contact with the chest-wall (the diaphragm alone intervening) about the level of the sixth rib. The lower margin of the liver usually corresponds with the free border of the ribs. In the line of the axillæ, the upper margin of the liver reaches to the seventh rib, and extends to the posterior surface of the chest to the lower margin of the tenth rib. On account of the possible elevation of the liver by conditions of the abdominal cavity—tympanitic distention, dropsy, abdominal tumors, etc., it is well to form an idea of the dimensions of the liver expressed in figures. Anteriorly, the region of dullness and flatness includes a distance of about three inches in the line of the right nipple. Laterally, in the axillæ, it includes a space measured by about four inches. Posteriorly, the measurement is from two to two and a half inches.

Percussion of the liver should be made both in the erect and recumbent positions. It is much easier to define the transition anteriorly, from external dullness to the tympany of the abdominal region, when the patient assumes the recumbent posture, since the abdominal walls become more flexible. The thick abdominal walls possessed by some effectually preclude an easy recognition of the tympanitic resonance, and might lead the incautious to infer enlargement of the liver when none exists.

In percussion, place the finger used as a pleximeter parallel with the ribs, commencing, we will say, at the third interspace. Percuss downward until absolute flatness is noted, even in forcible percussion; then percuss more gently upward, noting meanwhile that the resonance becomes more and more clear as one ascends the chest, until unmixed pulmonary resonance is determined. Unless there is disease of the pleural cavity, or the base of the right lung, the boundaries of the liver can easily be defined. Frerichs has given us a rule which is very useful in the diagnosis of these cases: The liver dullness can always be altered by a full inspiration; but if there is a pleural effusion, or chronic pneumonia, the level of impaired resonance or dullness is not changed during respiration.

The spleen occupies the upper portion of the left hypochondriac region. It is about four inches long, reaching from the free border of the ribs up to the ninth rib. It is about three inches wide, its anterior border lying in contact with the stomach and colon. Its convex surface is closely opposed to the concavity of the diaphragm, so that it influences the results of percussion in this region over nearly the entire extent of the organ.

The stomach varies widely in its dimensions and relations, under different circumstances, in the same individual. When moderately distended with gas it is comparatively easy to determine its boundaries and position. The cardiac orifice corresponds to the inner extremity of the seventh left rib, while the pylorus is found near a line drawn from the right nipple to the umbilicus, a little below the lower border of the liver.

The left or posterior border of the organ is bounded by the spleen; the lower border is usually nearly on a line with the umbilicus. The upper border is in contact with the under surface of the diaphragm and liver.

The colon is divided into four parts, the ascending, descending, and the sigmoid flexure. For our present purposes we must bear in mind the distribution of the three parts. The cæcum is situated in the right iliac fossa, immediately behind the abdominal wall; the ascending colon is smaller, and passes upward to the under surface of the liver, on the right of the gall bladder, where it bends abruptly to the left, and in this part of its course is spoken of as the transverse colon. The ascending colon is in relation in front with the convolution of the ileum and the abdominal walls; behind, it lies on the quadratus lumborem muscle and right kidney. The transverse colon is in relation by its upper surface with the liver and the gall bladder, the greater curvature of the stomach, and the lower end of the spleen, by its under surface with the small intestines, by its anterior surface with the anterior layers of the great omentum, and the abdominal parietes. The descending is more deeply placed than the ascending colon, and is smaller in calibre. Its relations are similar to those of the ascending colon.

The loops of the small intestines are surrounded, as it were, by the distribution of the colon. Normally they occupy a central position in the abdominal cavity. They are, however, loosely confined by thin mesenteric attachment, a fact of no little importance to the diagnostician, since they can be displaced in various directions by accumulation of fluid, or tumors occurring in the abdominal cavity. Percussion of the abdominal region should be made when the abdominal parietes are relaxed, if this be practicable, since the tone of vibration pertaining to each of these cavities can be more readily distinguished. The percussion note over the stomach, colon, and small intestine are alike in quality, viz., tympanitic, but vary in pitch.

The tympanitic resonance of the stomach is much lower in pitch than the resonance over the colon, and the resonance of the colon is lower-pitched than the tubular or high-pitched tympanitic resonance of the small intestine. The reasons for this variation are elsewhere stated.

The kidneys are situated in the lumbar regions, in the space corresponding to the two lower dorsal, and the two upper lumbar vertebræ; the right is a little lower than the left. Superficially, they extend from near the eleventh rib to the crest of the ilium. The right is bounded above by the posterior and inferior portion of the right lobe of the liver; below, by the cæcum; anteriorly, by the ascending colon, and posteriorly by the diaphragm and quadratus lumborem muscles. The left is bounded above by the spleen; anteriorly and inferiorly by the colon, and posteriorly by the diaphragm and quadratus lumborem muscle. These organs are so deeply situated, and so surrounded by muscular structures, that, unless they are enlarged, it is difficult to demonstrate their presence satisfactorily by percussion. Having thus briefly indicated the normal anatomical position of the thoracic organs and of the abdominal viscera, whose relations with them influence the result of physical explorations, we must also indicate a few of the more important of the vast number of changes within the abdominal cavity which influence physical diagnosis.

Influence of some abnormal abdominal conditions upon the thoracic viscera.—A moment's reflection will convince one that distention of any part of the intestinal tube with gas will tend to elevate the diaphragm and materially displace the heart, liver, and spleen. The stomach is capable of enormous distention by gas, especially whenever its pyloric orifice is seriously stenosed. The cardiac extremity can arise so high as the sixth or seventh rib in the left axilla. A ready means of identification is practiced by causing the patient to drink slowly a glass of water—the metallic tinkle of the fluid as it drops into the viscus is very perceptible. This method is very useful to differentiate this distention of the stomach from pneumothorax, or emphysema* of the left side.

Besides the displacements of the intra-thoracic viscera, the distention is capable of abrogating the diaphragmatic movements sufficiently to seriously modify the respiratory murmur. The pitch, quality, and rhythm of the murmur can all be influenced; but principally the last two attributes. The lungs may be so compressed that the quality of the respiratory murmur may become concentrated, harsh, or broncho-vesicular, and the pitch elevated, especially in the upper anterior and posterior portions of the chest, and the rhythm can be changed so that we hear only inspiratory movement of respiration; or the breathing may be interrupted in rhythm. In other instances, the quality becomes feeble, and the pitch very low. The displacements of the heart may embarrass its revolution, and cause modifications of rhythm, which it would be tedious to describe in detail. Even murmurs can be temporarily suppressed. Friction sounds, both pericardial or pleural, may temporarily vanish, by reason of extreme gaseous distention of the abdomen.

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* *See section* on Pneumothorax, and on Emphysema.

Dyspnœa is naturally a most important and frequent symptom, since the aeration of the blood cannot be effectively performed. This symptom developed from the above cause often demands our attention when we are called to treat various forms of pulmonary or cardiac disease, notably emphysema, or bronchitis in the former, or valvular disease in the latter case; the gravity of the case being lessened if the tympanites is relieved. Large abdominal effusions are capable of producing much the same series of deviations from the physiological, or morbid physiological status of the thoracic organs. Tumors in the abdominal cavity are mechanically capable of producing the same series of symptoms to a greater or less degree. A physiological example of these influences may be cited in pregnancy. In the male the inconvenience is observed more markedly in the respiratory organs than in the female, since the former are less capable of exaggerated superior costal respiration. Acute or chronic lesions of the peritoneal membrane are attended with deficient play of the diaphragm, and more or less modification of the respiratory movements. We are now prepared to proceed to the consideration of the special subjects of this handbook, the diagnosis of diseases of the heart and lungs.

PHYSICAL DIAGNOSIS.

PART I.

CHAPTER I.

METHODS OF DIAGNOSIS.

The most useful methods of physical examination are by means of percussion and auscultation. The other methods include palpation, inspection and mensuration, but are auxiliary to the first named. Let me describe briefly the percussion and auscultation of the healthy chest, as preliminary to the study of pathological conditions. In the first place, the position of the patient should be standing, if the most accurate results are to be gained; next to this the sitting posture, with the arms so placed as to render moderately tense the tissues covering the chest. If the abdominal region is to be studied the percussion might be made while the patient is lying on the back, or standing, so that the resonance obtained by percussion, while the planes of the tissues are in varying states of tension, may be attentively studied and compared. The best instruments for performing the act of percussion are the hands, the fingers of one hand being used as a plexor, those of the other as a pleximeter. The finger used as the pleximeter should be the fore-finger, but the middle or ring-fingers should be used when it is deemed desirable to percuss in succession several parallel planes of the chest. The fingers should be applied so as to firmly compress the various layers of tissue forming the walls of the chest or abdomen into one compact layer; the pitch of percussion is much modified by neglect of this practice, and mistakes are rendered possible, especially in the more disseminated and moderate pathological changes.

Another value of the digital pleximeter is that by it we are enabled to appreciate the amount of resilience or elasticity of the tissues percussed. Piorry was wont to remark that he felt the modifications of percussion resonance. Always observe, then, the impression of flaccidity, or tension of the tissue percussed. Passing now to the use of the plexor, we employ one or two fingers of one hand, which should be placed in such relation to each other that the tips are of equal length. Percussion may at times be made with one finger only; this is especially the case when it is desired to elicit but a moderately intense sound, but if resonance is to be developed from deep planes of tissue, then both fingers should constitute the hammer. The stroke itself should be delivered from the wrist, and by the impulse of the hand alone.

The secret of percussion, as in piano playing, is to encourage flexibility of the wrist, and as the execution of the pianist lies in the touch, which is secondary to a trained freedom of wrist movement, so touch in percussion depends on a similar training of the wrist, not on manual force. The knack of percussion lies in the ability to develop full vibration in resonant tissues, not in the forcefulness of the blow. I have dwelt upon the method of percussion, because the essential properties of resonance are profoundly affected by it, viz., quality or tone, and pitch.

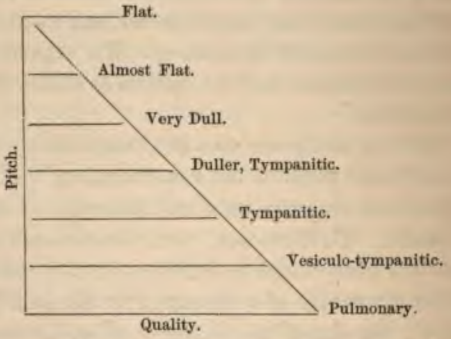
Quality of resonance depends quite as much on the execution, as on the state of the tissues. 1st. Quality depends very much on the order with which the waves of air follow rhythmically on one another; 2d. Their repetition at regular intervals, and with regular rapidity. It has been found by experiment that the number of waves must not be fewer than forty in a minute; otherwise they do not blend into a tone; *vice versa*, tone is influenced by their too great rapidity. The pitch of a sound depends on the swiftness with which the periodic waves follow each other, and in the number of shocks following each other in a given space of time; the swifter the succession of both, the higher the pitch. It is for this reason that faulty percussion is capable of developing sounds which lead to erroneous results. Percussion with both rapid and slowly repeated shocks must be compared; those given with medium rapidity are usually the most satisfactory. In the selection of a site on the chest for percussion, if possible, choose the interspaces in the chest, rather than the bony ridges; the alone caution we advise is that care must be taken to compare site with site on the opposing sides of the chest. It may be necessary to percuss over bones at times, and we propose to indicate the additional cautions to be observed. It is advisable, when possible, to percuss placing the pleximeter parallel with the axis of the ribs; exceptionally it may be necessary to place the pleximeter perpendicularly to the axis of the body. We are now in a position to advance to the study of resonance, which it is the function of percussion to develop. Light and darkness are understood by contrast; first fundamentally, then as the trained vision is exercised, shades are differentiated, and stamped as distinct. So the word resonance is a cardinal term indicative of reverberation, to be contrasted with deadness, or such complete absence of musical quality that a sound comes to be termed flat or toneless. We are accustomed to the term as applied to familiar objects capable of reverberation. Before describing the reverberation of the percussion of the human lungs, let us analyze the term resonance abstractly, to determine its accurate definition. Considered by exclusion, it is readily noted that it does not consist fundamentally in loudness; this is an incidental attribute of resonance, depending largely on the forcefulness of the motor impulse generating it and on the condition of the tissues encasing the lungs. In general terms I would say that intensity is not a factor in the integral composition of either resonance or the respiratory murmur. The duration of resonance is much dependent on the intensity of sound, and must also be laid aside in the inquiry as to the entity of resonance. The two properties germane to resonance are pitch and quality. Pitch being sponsor for the tone, quality sponsor for the classification of the reverberation.

As anatomists we are familiar with the structure of the lungs; as diagnosticians we invoke the conception of air confined in a multitude of sacculations, that is, the vesicles and lobules of the lungs; the trachea and bronchial tubes forming a tree, the branches terminating in the finest bronchioles. The essence of the subject is, that in those parts of the chest chiefly formed of elastic tissue the contained air is in a condition of moderate tension.

The bronchial tissue is more dense, less elastic, the contained air is confined within a more limited space, and its state of tension is more decided. The above paragraphs indicate that in the resonance called pulmonary the dominant ideas are elasticity, and consequently varying tension. To go a step further: we all understand the stomach and intestinal canal to be an air containing tube, of varying diameter, and its walls sustaining varying degrees of distention. We regard the reverberation developed over the system as hollow or drum-like, tympanitic.

Now we easily see two circumstances preside over the quality and pitch of the reverberation, viz., the varying size of the digestive tube and the state of distention of its walls. To illustrate: over the stomach it is manifest that the reverberation develops in the mind a sense of an empty cavity of some size, over the small intestine or colon; the size of tube being smaller, the reverse impression is gained. But a few moments' practice will convince any one that over the stomach, if fully distended with gas, the sound is much more acute, as compared with the graver note of the less distended viscus, and this irrespective of the size of the organ. Similarly the distended small intestine and colon yield a more acute note on percussion than the graver tone of the less distended state. The tension and elasticity of the walls of the digestive tube indicate that the vibrations of the air, if percussion is made, will be shorter in proportion as the tension is increased and elasticity diminished, and the pitch will be higher; just the opposite will occur if the reverse condition obtains. One step further—if percussion is practiced over a solid viscus, such as the liver, it must yield a toneless sound, that, in common parlance, would be termed flat or non-resonant, since its structure is not air containing.

To represent this diagramatically let me erect this figure of a triangle, which can be filled in as we proceed:—



To understand the term pulmonary resonance, which must become a typical sound to our ears, let us fancy air contained, as in the vesicular and bronchial tissues of the lungs, versus air distributed in the digestive tube; have you not at once the mental picture of a sound which is less tympanitic in quality, because less hollow in quality; less hollow because the air is distributed in smaller amounts in any given district; more low pitched because the wave of vibration of the air after impulse is longer, since the tissues are more elastic and the air is under lower state of tension. Behold! the terms quality and pitch assume definiteness, and we incorporate into our understanding a word painting of a sound of pulmonary quality and low pitch, which, once heard, becomes a reality no more arbitrarily than the empty tin trumpet resonance of our childhood is determined. But the stage of the respiratory act must modify the percussion resonance; we can cope with this idea of the influence on sound by a conception of the effect of a full forced inspiration upon the normal pulmonary structures. The amount of air contained within the chest and the capacity of the air sacs is increased; the tension of the walls of the vesicular pulmonary tissue notably exaggerated, while the elasticity is impaired, as the outcome of the percussion resonance departs from the typical and approaches the tympanitic; but since it partakes of the qualities both of pulmonary resonance and the tympanitic, it has been most justly termed vesiculo-tympanitic, or a sound of more high pitch than good pulmonary resonance, more tympanitic in quality, but still retaining sufficiently the qualities of normal vesicular resonance, to deserve its christening as vesiculo-tympanitic resonance.

To clinch this idea, so fundamental, we would refer to the percussion resonance as it exists in supplemental action of a single lung from disablement of its fellow, and to the state of substantive emphysema of the lungs.

With these three results of percussion, the flat, the tympanitic, the normal pulmonary resonance, and its modification, the vesiculo-tympanitic, the next step must be to consider the distribution of resonance in the physiological chest, and the influence of special conditions of the chest walls. The ability to map out the organs contained within the chest is one of the final steps in the study of percussion; it will suffice, at this point, to say that the most typical pulmonary resonance can be elicited over the upper anterior portion of the chest, above the third rib, in the axillary regions, and posteriorly below the angles of the scapulæ, the best resonance being located in the regions in the order as named.

Influence of Tissues on Resonance.—In the other regions of the thorax, it is evident that the layer of bones and muscles must seriously impair the clearness of resonance, as, for instance, in the region occupied by the scapulæ, and the thick muscles of the back, and in those cases in which the distribution of adipose tissue is unusually large. It can readily be imagined that the intensity of the sound must be the attribute of resonance, mostly modified incidentally; the pitch and quality may be dull, or almost flat. In the pathological conditions of the pulmonary tissue the intensity is of insignificant importance, and the quality and pitch loom up into primal prominence. The actual note of physiological resonance must be demonstrated and stamped upon the auditory sense. It is enough if these words have clearly indicated that resonance may be muffled, just as any other resonant body would be affected by similar conditions. Quite in contrast, we are frequently confronted by individuals who are very thin by constitutional habit; with others who have become thin through the atrophic changes in the tissues incidental to disease or advanced age; in all these cases the resonance is often of the vesiculo-tympanitic type. In childhood the bony thoracic walls are slight, and the integument and muscles are distributed in less considerable layers.

On this account we are able to develop more easily vibrations in the air contained in the lungs. In children the bronchial tissue predominates, the air is contained in less resilient cavities, and the vibrations must be shorter; now, since the stroke impinges more immediately upon the pulmonary substance, the note is more high pitched, or vesiculo-tympanitic. The same conditions are obviously present in adults who are spare and subject to chronic bronchitis and atrophic emphysema.

Exceptions.—It occasionally happens, in the physiological chest, that the percussion resonance over the right apex verges toward the vesiculo-tympanitic, or, at least, is more high pitched than on the left side; it is less significant of pathological condition than a similar modification over the left apex. (*See* page 29.)

Respiratory percussion is a method which has been described by Dr. Da Costa, as sometimes very useful. It consists in practicing percussion, either at the moment of full inspiration, while the person examined holds the breath, or at the end of forced expiration.

Over the apices, percussion at the end of full inspiration yields a sound of higher pitch and vesiculo-tympanitic (fuller vesicular) quality. The usual disparity between the right and left sides is preserved, the pitch on the left side remaining lower than the right. Broadly speaking, this effect can be noted more or less positively in all the regions of the chest.

A held expiration diminishes the resonance, but in the writer's experience this method is most useful during the inspiratory movement. The method will be developed in the study of abnormal conditions.

Another valuable method, yielding useful data as to the state of the pulmonary tissue of an entire side, is percussion over the clavicle. Its results, in quality and tone, often supersede the necessity of further investigation—a point of importance when it becomes desirable to shorten an investigation.

Finally, in describing the locations of resonance, the ribs constitute the appropriate landmark, with the medium line of the sternum as a fixed dividing line between the two sides of the chest. This is of especial importance when we attempt to record for others the limits of abnormal resonance; and it will prevent mistakes to define the record by inches. We would also reiterate in this place a general principle presiding over physical diagnosis, viz., that each result of the practice of physical diagnosis is based on the comparative examination of the two sides of the chest in each individual case. This is especially an important precept in connection with auscultation and percussion. Questions of quality and pitch being relative terms, each case being an independent study and a law unto itself.

CHAPTER II.

AUSCULTATION.

By auscultation we study the respiratory murmur, or the passage of air through the bronchial tree, from the trachea to the finest of the terminal bronchioles, until it is lost in the vesicular structure. Physiologically speaking, it is a murmur separated into two movements of equal length, the one occurring during the inspiratory, the other during the expiratory act. Clinically speaking, it is a murmur with a movement of greatest duration during the inspiratory period, which may be diagramatically indicated by the numeral 5, a period of pause, indicated by the figure 1¾, and the period of expiration, represented by figures 3¼. But the expiratory murmur is longer over some regions of the chest than others; for instance, in those regions most liberally supplied with bronchial tissue, and in those persons who possess some of the physical conditions which combine to yield a vesiculo-tympanitic percussion. The key to the comprehension of what is called *rhythm* by writers on diagnosis, is to understand that the pause is simply the period of vesicular collapse, during which the air is passing into the bronchial tubes, but so noiselessly as to be inaudible to the ordinary observer, so that we hear only the exit of the air from the bronchial tubes, and call it the expiratory murmur, whereas, it is but a part of it. The respiratory murmur, therefore, presents for our study the movements of inspiration and expiration, and the *rhythm* with which they are performed. Equally, however, with percussion, the chief attributes of the murmur are quality and pitch. Let it be especially noted that intensity of murmur has a very limited value as a diagnostic peculiarity, since it is wholly dependent on the amount of air which can enter and find exit from the lungs. The breathing in quiet respiration may be so shallow and feeble that its elements may be much masked. When the respiration is deeper, so that it can be defined, we appraise it very largely by its quality and pitch. This principle of classification is the same upon which we define the sound of wind instruments in general. The duration of the respiratory murmur is best appreciated when studied under the title of *rhythm*, which is a valuable indication of the true meaning of the respiratory murmur, in special conditions.

In studying the respiratory murmur, then, we award to the quality the place of first importance, second the pitch, relegating to the question of rhythm a position of subordinate value.

There are three types of respiratory murmur in the physiological chest, viz.: the tubular type, represented by the sound as heard through a stethoscope placed over the trachea; the bronchial type, most distinctly appreciated in individuals who are thin, and is heard most characteristically between the scapulæ, the region principally occupied by bronchial tissues; the vesicular type, audible at the anterior and upper portions of the chest, in the axillæ, in the infra-scapular regions, names which indicate with sufficient precision the confines of the situations. The tubular type is a murmur of a very concentrated or reed-like quality, hence its name; and the pitch is necessarily high, since the tube in which the sound is generated is not a large one. Essentially the concentration and high pitch are the most important factors of this sound. The bronchial type is a natural derivative from the tubular; it differs only in being less concentrated, less high pitched, although its tubular quality remains. It is simply named bronchial, from the tissue in which it is produced; with equal propriety it might be called less pronounced tubular breathing. The diffusion of the sound and the lower pitch of bronchial as compared with tubular breathing are to be explained by the situation of the bronchial tubes, embedded in a vesicular tissue which acts as padding might, around a series of reeds of similar size.

The vesicular breathing, normal respiratory murmur, as it is termed, consists in a further modification of this type—the air issuing into the vesicular tissue expands it with a rustling sound, which is diffused, because the vesicular tissue constitutes the bulk of the lungs; it is low pitched, because the cavities receiving the air, though limited in size, are very numerous. This murmur, termed by writers breezy, is typically to express the lowest pitched and the least concentrated sound which the student of physical diagnosis has to observe. As a parallel sign by auscultation to the vesiculo-tympanitic percussion, we note that division of respiratory murmer called broncho-vesicular.

Primarily this is merely normal vesicular respiratory murmur plus exaggeration of both its elements. It is present when a lung has undertaken supplemental activity, the intensity of the sound being explained by the increase of the respiratory function of that side, and has been termed by some writers exaggerated breathing. We find very marked broncho-vesicular respiration in children, since the bronchial tissue exists in excess of the vesicular. Over the chests of individuals who are thin, for obvious reasons, the intensity is increased.

Perhaps one or two instances from the field of pathology may fasten this phase of murmur in our minds. Broncho-vesicular breathing, in addition to the above instances, is heard in certain forms of emphysema, especially the atrophic, and in special stages of consolidation, to be hereafter described.

The most important fact to remember at this stage of our study is that when broncho-vesicular breathing is associated with any process of condensation of vesicular tissue the pitch is *high*, and grows higher as the process of condensation progresses. This is in profound contrast to the low pitch in the above mentioned conditions.

The study of rhythm relates to the appreciation of the time of the respiratory revolution and the deviations from the standard.

The physiological rhythmic development of any of the respiratory sounds depends chiefly on the amount of air introduced into the chest; so that in healthy persons partial inflation will develop a respiratory murmur of atypical rhythm, because the expiration will be shortened. Disease presents many such instances: for example, in early stages of phthisis the respiratory murmur is pronounced; as the process advances less air is inspired, and feeble respiratory murmur often becomes an important evidence of the change.

Reversely considered, the preponderance of bronchial tissue in a lung, for any cause, or the nearness of the bronchial tissue to the ear of the listener, will combine to render the expiration more and more *distinct*, until it may become as clear and distinct as the inspiratory murmur. From what has just been said, it is evident that the rhythm of respiratory murmur depends, to a great extent, on the relation of the bronchial to the vesicular structure, and the thickness of the thoracic walls, and the amount of air respired.

Distribution of Respiratory Murmur.—Clinically we find inspiration is heard as a more prolonged murmur than expiration, over the apices and in the axillary regions—nearly the same ratio prevails in the infrascapular regions, but the expiratory sound is sometimes less distinct than in the first-named situations—between the scapulæ the bronchial tissue predominates, and the two movements of respiration give a respiratory murmur more nearly equal.

Exceptions.—Occasionally the quality, pitch and rhythm of the respiratory murmur are altered, so that over the right apex the pitch is higher and the quality less vesicular than over the left—the rhythm also changes, the expiration being somewhat more prolonged. No very adequate explanations have been proposed for these physical signs. It has been thought more common in the right-handed folk, but a better working rule will be to carefully investigate, by all the methods of physical diagnosis, whenever any pronounced difference between the two sides of the chest can be detected. Note if the habit of using the muscles of the side is influenced by occupation or business, and decide in favor of physiological variation only after a strict analysis, by exclusion of any pathological process.

Method.—Finally, as to the method of auscultation: usually it is desired to examine a considerable area of the chest. This is best accomplished by applying the unaided ear to the chest, which has previously been stripped to one or two coverings, over which the examiner can place a smooth soft towel. With both men and women, when accuracy of investigation is essential, this rule must be absolute. Silk or merino underwear, unstarched linen, or muslin garments, may be worn without interfering with the examination. But in all subacute or chronic cases, the satisfaction to the practitioner, of accurate knowledge, and the comfort to the patient of an assured opinion, are so obvious, that the trouble required for an examination will readily be yielded. The science of physical diagnosis has received its worst blows from the hands of hurried and partial, rather than unskilled investigations.

But, at times, it is desirable to auscult a limited area, or acute disease, may make changes of attitude necessary for examination by the ear alone, impossible. For this reason it is necessary also to become familiar with the use of some reliable stethoscope. None is better than that made of thin bell-metal—now to be procured of Mr. Gemrig, of this city. Its weight is 1 oz., and as a perfect conducting medium, it is unsurpassed by any with which we are acquainted. Certain other stethoscopes are vended, not so desirable, in the writer's opinion. They may be recognized by the thickness of the barrel of the instrument, the thickness of the ear-piece and the bowl-shaped expansion at the chest orifice; the one advised resembling a funnel. The binaural instrument, much used in New York, is a most convenient instrument; it can be adapted, by means of its flexible tube, to any position of the patient, and one of its best claims for use lies in this fact. The objection exists in the fact that it intensifies sounds, so that an untrue estimate may be affixed to them. Training will eliminate this objection, and one will do well to secure familiarity with its use; although we believe that the first named single-tubed instrument will be most satisfactory.

There is one other application of the analysis of the respiratory murmur, termed a study of *vocal resonance, and vocal fremitus*. When phonation is attempted, the consequent vibrations of air are carried downward as well as upward. The downward path conducts the waves of sound into the fine bronchioles, and thence into the vesicular structure of the lung; thence these sound waves, broken up of necessity into numerous fine vibrations, are transmitted to the chest walls, and can be appreciated by the ear, as the vocal resonance. The vocal fremitus is the same vibration demonstrated by the application of the hands to the bared chest, the method being designated as palpation. To confine the present remarks to auscultation, let me observe that the vocal resonance in physiological chests bears parallel relation to the analysis of respiratory murmur. In those situations at which vesicular respiratory murmur is located, the vocal resonance is a diffused, low-pitched vibration, differentiated elsewhere by the same terms as are useful in auscultation. Its quality is diffused, or concentrated, and the pitch varies in direct relation to the increased concentration of quality. We have said that intensity of sound is not an important factor of the breathing, indicating the lesions of condensation of pulmonary substance. In the region at which the respiratory murmur becomes bronchial, the vocal resonance is termed bronchophony, indicating a concentrated high-pitched sound; over the trachea the vocal resonance is more concentrated and higher in pitch, tubular vocal resonance. In harmony with what has been said, we observe, by way of illustration, that if vicarious respiration be carried on the vocal resonance is exaggerated, with the parallel signs of broncho-vesicular breathing, and vesiculo-tympanitic percussion.

In pathological states, the vocal resonance becomes concentrated, high pitched, meriting the term bronchial voice or bronchophony. The quality bearing direct relation to the degree of bronchial breathing, tubular breathing being accompanied by most marked bronchophony.

Variations.—The variations from the physiological standard are in identical comparative relation with the variations in the respiratory murmur; equivalent expressions are used to designate the varieties of vocal resonance, so that a recapitulation is not required.

Vocal Fremitus.—The application of vocal fremitus is more simple, since the fingers are incapable of appreciating the many variations of tone and quality which the ear can readily detect. The vibration of the voice can be felt more or less distinctly, as a tremor, which has been compared to the vibration felt over the back of a cat when the animal is purring, and has been termed a purring tremor.

Over those portions of the chest wall where the tissues are thinnest the vibrations are most distinct, naturally in the upper anterior portion of the chest, and the lateral regions, then posteriorly, below the scapulæ. Very marked difference in the amount of vocal fremitus on the respective sides of the chest anteriorly, has been considered pathological; although it has been agreed that the vocal fremitus is usually more distinct over the right apex than over the left. So many conditions influence the conduction of waves of sound in the chest, and since vocal fremitus depends on this conduction, it would appear safest to say that unless the variation is that of marked increase on one or other side, the evidence is not to be admitted as testimony of decided change in the textual condition of the lungs.

Naturally the individual timbre of the voice will much affect the vocal resonance and fremitus. The more sonorous sounds developing the most pronounced examples of vocal fremitus. Another precaution relates to the method of phonation. This should be practiced slowly; the numerals, one, two, three, or twenty-one, twenty-two, twenty-three, should be pronounced, so that the vibration developed by each note should be a distinct entity, and each vibration should cease before a second commences. The same caution is essential to the accuracy of the results of vocal resonance.

Whispered Voice.—In pathological states of the lung, especially those attended by formation of cavities, it will be found advantageous to study the whispered voice by auscultation, but the vibrations are too feeble to be appreciated in the normal condition of the lung tissue, either by auscultation or palpation.

Inspection and mensuration are methods of examination conveniently considered together, since they are usually concerned in the study of the same phenomena; they are the filling out or making more exact the results obtained by the other methods.

The most important result of mensuration is found in the satisfactory record which it can yield of the contour of the chest. This is accomplished by taking measurements of the chest at different sectional planes. The tracings can be made by applying to the chest strips of sheet lead, three-quarters of an inch wide and graduated along the border. The strips can be started from the spine, and end at the median line of the sternum, which point is accurately noted on the strips. It is easy to detach this metal strip by gentle manipulation without altering its curves. If a large sheet of paper be spread upon a table near at hand the strips can be laid upon it. A pencil is then drawn closely along its curve, a mark made to indicate the position of the spine and sternum, and then, on removing the strip, we have a precise tracing of the surface of the thorax measured. We can repeat this for both sides, being careful to place the spinal and sternal points at exactly corresponding points on the first tracing. Besides being a record of contour, this method is an accurate mensuration; we can note the relation between each diameter of the two sides. It is usual to take a tracing at a single level, choose the lines of the fifth or sixth intercostal space, or tracings at different levels can be made.

If it is desired to record the state of the chest, using full inspiration and strong expiration, and to ascertain the extent of the respiratory movements, it will be necessary, if this method be employed, to make a separate tracing of the chest at different states of respiration.

The graduated tape is a more rapid method, and, unless the contour of the chest is to be kept in record, it will be preferable. The tape is to be applied moderately tightly to the spine and the mid-sternal line around the chest, a mark having been made at the level designed to measure the chest. The number of inches registered by the measurement of the two sides are simply added together and the result read off. If we wish to measure the longitudinal diameter, a line from the clavicle to the base is selected. There may be a slight difference between the two sides of the chest, owing to the habit of the patient in using chiefly the right or left hand; a considerable difference is compatible with the healthy standards.

At full inspiration, the girth of the chest can be increased from two to three inches, a fact of which the method of measurement just described takes cognizance.

Inspection is primarily of value in defining the ideas of size, shape, and movements. In size, the normal chest should be symmetrical in each of its parts, as well in shape as in movement. Its characteristics need no detailed description in this place. The difference between the two halves of the chest alluded to in mensuration prevail. Inspection gives a mental picture of departures from the standard outline, which have been classically defined in some peculiarities of shape; notable among these are the phthisical, the rachitic, and the emphysematous chests. In the *tuberculous thorax*, the vertical diameter of the chest is increased with marked obliquity of the ribs, and a corresponding shallowness in the antero-posterior diameter. The shallowness is especially marked in the upper portions of the chest, owing to the poor development of the external thoracic muscles, and the consequent imperfect expansion of the upper lobes of the lungs. The infraclavicular regions are deep and pronounced. The clavicles and shoulders prominent and overhanging. Partly in consequence of this and partly in consequence of the weakness of the trapezius and other muscles of the upper and back part of the chest, the scapulæ are tilted forward, and their angles project markedly. This appearance, when highly developed, well justifies the name given to this alteration of the chest, of "alar," "pterygoid," or winged.

A transverse section of such a thorax shows that the elliptical form is greatly intensified, the transverse diameter increased, the antero-posterior are shortened, with a depression corresponding to the external region. This increase in the vertical diameter is intensified by strong action of the diaphragm, which is obliged to contract powerfully, to atone for the insufficient respiratory movements of the upper lobes of the lungs. It is this deficiency, and small capacity of such chests, associated with general constitutional weakness, which predisposes persons with marked alar chests to pulmonary phthisis.

The "flat" chest is often seen independently of the "alar" chest. It may be regarded as indicating a predisposition, or phthisical tendency. It depends upon a change in the shape of the cartilages of the true ribs, which lose their curve, and become straight. The sectional area of such a chest is much changed, and its vital capacity diminished. These alterations of the form of the chest appear to be congenital in many *instances,* and mark the manner in which, by the transmission of peculiarities of physical conformation, a form of inherited tendency to disease is brought about.

In other cases they are acquired, or at least greatly intensified, owing to imperfect expansion of certain portions of the lungs during childhood. The facts are practical, and teach a lesson that care in reference to providing suitable gymnastics for children should be observed.

The Rachitic Chest. The Pigeon Breast.—These modifications may be considered together, since a study of the same phenomena explains both conditions.

Rickets is a disease of infancy and early life, one of the principal features representing a softening of the ribs and their cartilages. The latter represent a less specialized state of connective tissue; manifestly, then, the softest parts of the ribs are near their sterno-costal articulations.

When the diaphragm descends during inspiration, the softened ribs are not able to maintain the distention of the chest until a fresh supply of air enters, at the next inspiration. Consequently, the ribs yield at the costo-sternal articulation, being drawn inward, until a shallow longitudinal groove is formed along the side of the chest, parallel, or a little external to the sternum. Now, if the deformity is uncomplicated by frequent catarrhs, these changes tend to disappear to a remarkable degree as the health improves. But the imperfect rarification of the air contained in the lungs promotes pulmonary congestion, and this in its turn predisposes to bronchial catarrh, the sequel of which is the pigeon breast, more or less trace of which can be found whenever the rachitic state is prolonged.

Pigeon Breast.—The essential character of the chest is a straightening of the true ribs, which throws the sternum forward, the transverse diameter of the chest becomes narrower, and the appearance is prow-shaped. Rachitis, defective inspiration occasioned by chronic catarrhs, enlarged tonsils, etc., have been mentioned as predisposing causes.

The modifications of *emphysema* are noticed in the chapter on that disease. In studying the *movements* of the chest, there should be both superior and inferior thoracic expansion and retraction, but there should be some diaphragmatic breathing, more marked in the male than in the female. In the latter the superior mobility of the upper ribs is physiological, and obviates the necessity for as much diaphragmatic movement as in the male. In inspection, as in all physical examinations, the deviations are not to be compared to an ideal standard theory, but carefully contrast each lateral half with corresponding parts of the chest in the *same individual.* Carefully note in detail subclavicular space with subclavicular space, axillary region with axillary region, etc.

Practice inspection with the body in the easiest and most natural position, sitting or standing, with the surface exposed or covered only by a thin, tight-fitting garment.

Inspection properly employed becomes a most useful diagnostic method; further deviations from the ideal or individual standard will be indicated in appropriate sections.

CHAPTER III.

PRINCIPLES OF CLASSIFICATION. CROUPOUS AND CATARRHAL PNEUMONIAS.

The object of physical diagnosis applied to the lungs is to ascertain the textural conditions of the pulmonary substance. The analysis of cases by this means might properly be designated the local diagnosis; which in some instances may be sufficient to establish what might be termed the phase of the pathological process going on in the system. But in an equal or greater number of cases, a study of the etiology and symptomatology must be combined, to establish a complete comprehension of any process. The neglect of this method of study has discouraged many investigators in this direction, and has dwarfed the growth and development of this cardinal aid in the investigation and correct appreciation of disease. For purposes of clinical study the disease of the pulmonary parenchyma may be grouped in three classes:

1. Those in which the essential pathology consists in an alteration in the vesicular pulmonary structure, sometimes with a moderate or inconsiderable alteration in the nature of the inter-alveolar substance and the alveolar walls.

2. Those conditions which involve special changes in the inter-vesicular structure, with inconsiderable involvement of the vesicular wall and bronchial tissue.

3. Those cases in which the principal pathological

alteration occurs in the bronchial tubes, with but moderate structural alterations in the vesicular or inter-vesicular tissue.

This classification includes both acute and chronic pathological processes, but applies chiefly to those of chronic type.

In the acute forms, we have very often two of the tissues above named involved, in distinctly designated diseases; in which the process in each tissue, the bronchial and the vesicular, is separate.

We allude to morbid physiological changes designated acute croupous or catarrhal pneumonias, on the one hand, and to acute inflammations of the bronchial mucous membrane on the other. As illustrations of the chronic processes we have in the first group the different forms of catarrhal inflammation of the lungs, commonly known as chronic phthisis, but which are sometimes the result of both acute croupous and catarrhal pneumonia.

We also include the varieties of emphysema, as important lesions.

In the *second* group belong those forms of interstitial inflammations of the lungs originating from specific or other causes, commonly known as fibroid phthisis. In the *third* group, those forms of inflammation in which the bronchial tissue is specially involved, termed broncho-pneumonia, usually preceded by repeated colds, and often manifested merely as bronchitis, for years. It is a condition associated with various *slight* tissue changes, usually in both lungs; but which terminates in that textural *loss of substance* in one or both lungs which has led to the definition *broncho-pneumonic phthisis*. This is *to* be understood as merely a diagramatic representation, intended to facilitate clinical study, and although we have laid down these conditions as occurring separately, in reality, these forms so interlace that, practically, they are often difficult to distinguish.

Pneumonia Percussion.—Let us consider, in the first place, the evidence by physical diagnosis of the filling up of the vesicular structure of the lung in acute croupous pneumonia.

The physical signs of this condition are usually described as most marked in what is called the second stage; in reality, this stage represents the malady at its full height. We will subsequently study acute catarrhal pneumonia, which is often synonymous with rapidly developed phthisis. Croupous pneumonia is commonly described as lobar, although it may involve only part of a lobe; yet there is a strong tendency for the disease to spread throughout the entire lobe. The real distinction between this process and that of acute catarrhal pneumonia rests on the pathology. In the croupous process the exudation is poured out *into the vesicles*, which are the only structural parts of the lung primarily involved[*].

In croupous pneumonia the lower lobe of one of the lungs is usually involved, but it may affect the entire lung upon one side, or any of the lobes separately. In contrast, catarrhal pneumonia is usually distributed in the upper lobes in one or the other side of the chest, but may involve areas or patches distributed through the middle or lower lobes. This characteristic is the result of its parentage, viz., a descending bronchitis. The acute varieties will be separately considered, its chronic forms under the title of phthisis.[†]

[*] *See* Catarrhal Pneumonia. [†] Increasing Consolidation. Phthisis.

We wish to describe in this place the physical signs of a pathological condition in which each lobule in the affected district is completely filled with an inflammatory new formation, only the bronchial tissue remaining pervious to air. (*See* illustration.)

Two of the natural conditions of pulmonary substance are, violated elasticity and vesicular air-containing capacity; the bronchial tubes, however, usually contain air, but the air is confined under an abnormal tension. Two changes necessarily result in the percussion note, to wit: its quality becomes *very dull*, and the pitch *very high*, since the only air from which resonance can be developed is contained in the bronchial tubes, surrounded by the dense consolidation. The percussion note is rarely flat, as in pleural effusions, since some air is mostly passing in and out of the bronchial tree.*

Deviations.—In exceptional cases so much pressure is exercised by the new formation upon the bronchial tubes, that they become compressed, collapse, and little or no air being permitted to enter the affected region, the percussion becomes flat.

It may happen in pneumonia, especially of the upper lobes, that percussion resonance is neither flat nor very dull; but instead of this, the vesicular or subtympanitic sound, as already described, is developed. The cases in which this resonance occurs are those examined early, before the stage of total consolidation is reached. In these cases the first effect of the inflammation is to impair the elasticity of the lungs; its ability to empty itself of air in expiration is crippled, and at each effort at inspiration the distention of the vesicle is somewhat increased. The sound has been ascribed to the air confined in the bronchial tree in its upper divisions, the resonance being favored by some unusual distribution or calibre of the tubes, or some unusual thinness of the thoracic walls, rendering vibration of air in the bronchial tubes possible. In most cases the first explanation is the only defensible position, since the subtympanitic sound is transient, it is almost always succeeded by the usual very dull percussion sound. (*See* Respiratory Percussion).

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* *Percussion* in initial and terminal stages. See page 63.

Auscultation.—In consolidation of the lungs in pneumonia or any other rapid form of alteration from a physiological to a morbid state, the respiratory murmur is nearly always an intensely pronounced sound, in notable contrast with all the chronic consolidations of the lungs in which the intensity or volume of the respiratory murmur is feeble. The obvious reason is that in case of chronic consolidation, the lung involved is gradually disabled, and slowly but surely the air is diverted to those portions of the pulmonary substance in which it can be most thoroughly aerated, and less and less is distributed to those parts incapable of functional activity. The more important attribute of the respiratory murmur, viz., the quality, now claims our notice. It is readily understood that this must be tubular, since the bronchial tissue is the only tissue permeable to air in the affected district; the pitch is also high. The typical tubular breathing is so concentrated as to develop the impression that the air is rushing in and out just beneath the ear. When the consolidation is complete, the echo is brazen or ringing, as though the air were vibrating through metal tubes. The rhythm, the *third attribute* of the respiratory murmur, must be a come-and-go murmur, of equal length in the inspiratory and the expiratory movements.*

Deviations.—The first deviation relates to rhythm. The breathing is none the less tubular or intensely bronchial, although only inspiration can be detected. Hence the importance of appreciating the quality and pitch of respiration as fundamental. It is often the case that when pneumonias pass into the third stage, and resolution is delayed, one can hear only the inspiratory movement, and the longer the consolidation persists the more likely is this to be the case.

The second deviation relates to intensity, and is subject to the same rule; the longer the consolidation persists, the less intense is the respiratory murmur, since, naturally, the air is more abundantly distributed to those portions of the tissue best adapted to oxygenate it.

The Vocal Resonance.—Having ascertained the quality of the respiratory murmur, the vocal resonance is readily classified. The sound is termed bronchophony, but it may be so concentrated in quality and so high pitched as to be termed tubular bronchophony. If the timbre of the voice is very high, or the bronchial tubes are compressed by exudation, transmission of the waves of vibration after phonation is not appreciable.

Vocal Fremitus.—As a rule, this sign is available in this and all other cases of vesicular consolidation. It is increased in all typical cases; viz., those in which consolidation is equally distributed throughout a section of lung. And yet, on the one hand, the timbre of the voice, the thickness or thinness of the chest walls, and, on the other hand, the fact that sometimes, owing to *the large amount* of air entering the unaffected portions of the lungs, the volume of vibrations are distributed in such ample waves to these healthy portions that the combined result is confusing. The regions over which the vibrations are distributed with maximum intensity in the normal state of the lungs, must, of course, be kept in mind while making these observations.

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* *Râles in Croupous Pneumonia.* See chapter on râles

Summary.—We have as typical signs of the complete consolidation of a district of lung tissue, very marked dullness on percussion, tubular breathing, tubular bronchophony, and increased vocal fremitus. In children who possess very thin chest walls these physical signs are even more marked. For differential diagnosis between croupous pneumonia and pleurisy, *see* section on pleurisy.*

Catarrhal Pneumonia, Acute.—The distinction in the physical signs from croupous pneumonia depends upon the recollection of the fact that while in croupous pneumonia the radicles of the pulmonary artery are chiefly the source of the inflammatory exudation into the vesicles, in catarrhal pneumonia the radicles of the *bronchial arteries* are chiefly at fault. The essence of the disease consists in the intertwining of descending bronchial catarrh with thickening of the vesicular wall and inter-vesicular substance, but with only a *partial occlusion of the vesicle*, with cellular proliferation, composed of leucocytes, corpuscles from the inter-vesicular connective tissue, together with epithelial proliferation.† It is also associated with atelectasis. The vesicles not being absolutely occluded, remain somewhat pervious to air, and the process itself is *diffused* over the lungs, although there is a special tendency to involve the apices. Since, then, an entire lobe is not involved, absolute solidification does not occur as a rule, unless over a small area, while croupous pneumonia, nearly always involves a lobe, more or less completely. The elasticity of the lung is, however, seriously modified, the tension of the contained air is increased, and the result is elevation of percussion pitch, with usually a dull quality, proportionate to the extent of the process; but the diffusion of the consolidation, and the intervening over distended vesicles may render the quality somewhat tympanitic. It is never flat.

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* The differential diagnosis includes pleurisy with effusion, pleuro-pneumonia, and the forms of catarrhal pneumonia. *See* appropriate sections.

† *See Illustration.*

Auscultation.—This closely corresponds. The respiratory murmur becomes bronchial, or broncho-vesicular, according to the proximity of the areas of disease. This is in strong contrast with the tubular brazen murmur heard in the fully developed croupous process.

Fine subcrepitant or small mucous râles will invariably be heard over a widespread area *during the course of the disease*, as the evidence of associated capillary bronchitis.

In the croupous process, the initial stage only is marked by the crepitant râle, and the stage of resolution by a subcrepitant râle, due to the softening of the exudation.

But the evidence, so far as the study of the râles is concerned, is incomplete; we must recollect that the invasion of croupous pneumonia is sudden, its distribution is lobar, and the subcrepitant râles occur when resolution begins, some days after the inauguration of the process.

In the catarrhal form the râles are a marked feature from the first, since the parentage of catarrhal pneumonia is bronchitis.

While all this is true, even in lobar pneumonia, an accumulation of mucus in the finer bronchial tubes may give rise to subcrepitant râles. After an analysis of the physical sings by auscultation and percussion, we would prefer to unite the evidence deduced from a study of the clinical history, including especially duration and etiology.

Vocal fremitus and resonance confórm to the rule that the more dense the consolidation, the more pronounced the vocal resonance and fremitus.

We would nucleate the facts relating to the *subsequent history, first of catarrhal, and then of croupous pneumonia.*

Catarrhal pneumonia pursues a slower course than croupous pneumonia, and the physical signs disappear very gradually. The febrile phenomena disappear slowly with the resolution. On the other hand the process may advance until the vessels become more and more choked with cells, the radicles of the bronchial arteries are compressed, the circulation is interfered with, necrosis is induced, and in adults, very commonly, a slow or rapid phthisis results. The same may happen in children, but it is unfrequent; commonly after attacks of catarrhal pneumonia a tendency to recurrence of bronchitis may ensue.*

In *croupous pneumonia* resolution is more common in from five to ten days. The exudation disappears, partly by absorption, partly by expectoration, through the medium of a rapid fatty degeneration. If, however, this favorable change does not occur, the resolution may be delayed, and yet occur after the lapse of two or three months, meanwhile the lung remaining in a state of gray hepatization. But more frequently the tissue may pass into the state of purulent infiltration. The tongue will be more or less dry, the fever of hectic type sets in, recovery may occur, but death from exhaustion is more common. Exceptionally, the formation of excavations of the lung complicates this stage, which then becomes an unusual form of phthisis, to be diagnosticated chiefly by the history.

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* See Capillary Bronchitis. Catarrhal pneumonia followed by resolution is mostly observed in children; this is possible but improbable in adults.

The physical signs of *initial and terminal stages* of these pneumonias, produced by the changeful conditions in the lungs, are represented in many of the different stages of the development of subacute and chronic phthisis. It is our plan to represent the varying states of elasticity and intra-pulmonary atmospheric tension as affected by pathological processes in general. The above conditions will, therefore, be included later, in the hope that a broad view of the meaning of physical diagnosis will be obtained.

There are two signs that will be introduced here, which belong essentially to the initial stage of croupous pneumonia; one is the disappearance of the chlorides simultaneously with the advent of the process, and the other is the symptom of hæmoptysis, which is not an essential phenomenon, but is a constant symptom when pneumonia sets in among the young or vigorous.

In acute processes, whenever the aerating surfaces are curtailed, dyspnœa is a marked symptom, but in subacute or chronic processes the supplemental action of the *uninjured pulmonary* tissue, or the diminished demand made by the system for oxygenated blood, owing to a reduction of the systemic vital activity, result in removing this symptom from consideration. Or, if dyspnœa remain a symptom it is connected with exertion upon the part of the patient. Again, in all acute pneumonic pulmonary processes a dark red flush may be seen upon one of the cheeks, frequently but not invariably on the affected side.

CHAPTER IV.

SUBACUTE AND CHRONIC CONSOLIDATION.

Leaving the various forms of bronchitis and emphysema to be described subsequently, we now pass on to the consideration of the subacute and chronic consolidations.

The classification and nomenclature of these processes is to be decided after a careful study of the combined etiology and symptomatology. This is a portion of the subject best appreciated in the immediate presence of patients, or must be deduced from studies of works devoted to the descriptions of the details of disease.

It is for us, while considering this part of the subject, to recall that although all the constituents of pulmonary tissue are involved, the lesions are confined chiefly to that constituent known as vesicular. Let us compare their diagnosis with the acute consolidations just described. To accomplish this it is necessary to review, by a bird's-eye glance, our knowledge of the distribution of these newly formed products of a morbid physiological action, and with these to group those non-inflammatory processes in the pulmonary substance represented by hypostatic congestion, atelectasis, etc. In all these conditions the air is prevented from freely entering districts of lung substance, and yet the vesicular tissue is not absolutely condensed. The following remarks, then, include with the *above those conditions* of consolidation which are partial or disseminated throughout the pulmonary substance, based on inflammatory action. The initial lesions of the various forms of phthisis are disseminated through portions of one or more lobes.

In the early stages of consolidation (incipient phthisis), permanent lesion first manifests itself by insignificant alterations, predominant either in the interstitial, the bronchial, or the vesicular tissue, and these changes advance by slow but constantly increasing encroachments upon the neighboring tissues, until, perhaps, the entire pulmonary substance is involved. But the forms of phthisis are best understood if an outline of their pathological anatomy (in connection with the subdivisions) is considered in detail.

(*a*) These lesions may consist in patches of consolidation in vesicles which are in close relation with extensive districts of nearly normal lung tissue. The important changes in the non-consolidated vesicles consist in a supplemental distention of the air sacs, which is the case if the phthisical process is not extensive (initial consolidation usually in the apices).

(*b*) Another of the most essential features of subacute or chronic alteration, in addition to consolidation of vesicles, consists in an infiltration of the vesicular wall, and an increase in and infiltration of interalveolar tissue; this process slowly but surely destroys the elasticity of the pulmonary substance, and at places where intervesicular consolidation has not kept pace with the changes in vesicular walls and interalveolar tissue, the vesicles become substantively emphysematous; that is to say, the vesicular septa become atrophied, and a lobule is composed of a single large vesicle; or the vesicular and interalveolar thickening can occur, although the vesicular septa are not atrophied.* Pathologically considered, cirrhosis of the lung is but an advance in the condition, and implies a grossly preponderating alteration in the intervesicular structure, although associated vesicular consolidation persists to a greater or less extent.†

(*c*) Finally, there are cases in which chronic bronchitis results in bronchial thickening, associated with an *atrophy* of some of the vesicular tissue, leaving dilated emphysematous air sacs, interspersed among the normal, and those in which partial atrophy (of intervesicular substance and intravesicular septa) had taken place. The pathological picture includes, perhaps, a small amount of cirrhosis, sometimes more or less vesicular consolidation, but always dilatation and thickening of the bronchial tubes. At times there may be a blending of bronchitis, with the vesicular changes described under section (*b*). (*See* Atrophic Emphysema.) In any one of these cases there is a permanent distention of more or less of the pulmonary substance, and the consequent loss of elasticity involves increased intra-pulmonary atmospheric tension, which prevents physiological inspiration and expiration, and favors an increased residual column of air in the lungs. In natural sequence follows imperfect aeration and its result, dyspnœa, and the multiplied consequences of deficient pulmonary oxidation. The bearing upon the physical examination is so potential that this picture must be kept constantly in view, especially since we shall find every form of subacute or chronic pulmonary *alteration* includes some of these processes.

**See Fibroid Phthisis.* †*See* Atrophic Emphysema and Bronchiectasis.

We have said that a study of etiology and symptomatology is essential to a full realization of the scope of physical diagnosis, besides being helpful in correctly interpreting the physical signs themselves. To illustrate the point of etiology: speaking generally, the rapidity or non-rapidity of the development of the inflammatory process bears direct relation to the character of the tissue change. The more acute the process, the more probable is it that predominant alterations will occur in the vesicular tissue, with actual vesicular solidification; the more chronic the process, the more likely one is to find the paramount alterations in the interalveolar or bronchial tissue, with less extensive areas of vesicular consolidation. Further, etiology teaches that the distribution of the changes in the bronchial tissue is the sequel of recurrent bronchial catarrh. The lesions are situated in both lungs, until ultimately the development of local changes in all these three divisions of pulmonary tissue removes the disease from the nomenclature of bronchitis and establishes the designation broncho-pneumonia, or peri-bronchial phthisis. As a point in differentiation, this form of phthisis is located at those portions of the lung most freely supplied with bronchial tissue, *i.e.*, the roots, etc. (*See* section on Bronchiectatic Cavities.)

Another example of the value of etiology is found in the analysis of cirrhosis or fibroid phthisis of the lung. We have said that fibroid phthisis is both unilateral and bilateral, and is connected with prior bronchitis; it is also sometimes an expression of the syphilitic taint. While this is the case, it is also true that if phthisis has originated, the syphilitic process may be *engrafted* on any of its stages, or any of its forms.

Again, it is equally true that the fibroid process may develop independently of either syphilis or bronchitis, by virtue of specific irritants incident to special avocations, or in some form of chronic phthisis or chronic pleurisy.

The manifestations of *syphilis in the lungs* may be summed up in this connection, as follows:—

The specific process sometimes participates in the more chronic forms of phthisis, especially with the fibroid or bronchial changes, although vesicular consolidation in varying amount may be co-existent. These lesions necessarily specially manifest themselves near the roots of the lungs, or the distribution of the larger bronchial tubes. Gummata may be produced anywhere in the intervesicular tissue, usually near the visceral pleura. They occur in the deeper layers of the costal pleura, or on the periosteum of the ribs. Owing to their peculiar anatomical formation their subsequent history is one of combined caseous and fatty degeneration. Fibroid development may ensure their adhesion to the visceral and costal pleura, and they may point externally, with or without many of the appearances of inflammation in adjacent tissues; or, they may open internally, resulting in the formation of cavities; or they may remain stationary for an indefinite period. The symptomatology may be protean in variety, usually it includes a more gradual advent and progress than in similar forms of phthisis, without the specific taint. We do not usually have much fever, but cough or expectoration are in proportion with concomitant bronchitis; still more rarely do we have hæmoptysis. In *diagnosis* physical examination is useless, in view of the above facts. The only certain means of diagnosis consists in observing the usual signs of the prior or co-existent specific systemic poisoning. We call especial attention to the thickening of the periosteum of the head of one or both clavicles, the substernal tenderness and the thickening of the tibial periosteum.

The lesions in the larynx produced by the specific changes in the cartilages result in imperfect phonation, from the necessarily incomplete approximation of the vocal cords. Special works on this subject must be consulted for further details.

Effects on the Pulmonary Tissues.—The various foregoing changes modify the pulmonary tissue as an air-containing organ, and may be summed up as follows: The normal elasticity of the pulmonary substance is reduced by the actual condensation or solidification in the tissues concerned. The air is encased under more or less marked abnormal tension, and the vital capacity of the lungs to contain air is decreased. The air channels and chambers are all diminished in size. When we wish to differentiate, we find the point of symptomatology illustrated in the clinical history.

For instance, the more chronic the process of inflammation within the chest, the more probable is it to result in the formation of imperfectly organized connective tissue. This tissue possesses contractile properties which result in very decided alterations in the contour of the chest; especially are these alterations manifested in chronic pleurisy and fibroid phthisis.

Again, catarrhal pneumonia is associated with a history of recurrent cough, extending over a period of years;

fibroid phthisis, with syphilis, etc. Thus, subjective and objective facts are corroborated, when their study is blended with the results of the analysis of the physical diagnosis.

Use of the word Phthisis.—After the preceding summary of the nature of phthisis, we are justified in often using the word as a generic term, indicating not only the various divisions of tissue change already described, but also similar conditions of pulmonary substance, brought about by diverse causes, among which hypostatic condensation, infarction with blood from any cause, atelectasis, may serve as illustrations.

Diagnosis of Early Phthisis.—*Percussion.*—The tissue alterations in early phthisis, so soon as they can be recognized with certainty, include changes of resonance noticeable in the pitch and quality. In the involved area the pitch of the percussion note is elevated as compared with other regions, especially with the corresponding half of the chest. The quality of the resonance verges more and more into the vesiculo-tympanitic type. Both these departures are dependent on loss of elasticity and increased tension in the pulmonary tissue in the involved district. Perfect expiration is largely dependent on the inherent resiliency of the lungs, and the loss of this function implies that in both stages of respiration more air is confined in the pulmonary district diseased than is normal.

Auscultation.—The variation from the standard is mainly in the direction of rhythm, but includes a change in the quality. The study of rhythm usually very soon reveals prolonged expiratory movement; the change in quality soon follows. The statement of two facts explains the conditions. First, pulmonary elasticity is diminished, vesicular collapse is, therefore, slower; and second, the bronchial portion of the expiratory act is much obscured by the slow vesicular collapse; consequently we note the result as prolonged expiration. Sometimes the rhythm is termed *interrupted or cog-wheeled*, because the modification of the elasticity of the lung prolongs the inspiratory and expiratory period so much that the breathing appears interrupted, jerking, or cog-wheeled.

More inspiratory force is required than pertains to a single effort at inspiration, and therefore, more than one effort must be made before complete inflation of the lungs occurs; and similarly the expiration is accomplished in a spasmodic manner.

Prolonged expiration may also be due to a loss of consolidation pulmonary elasticity, in cases of supplemented breathing or in emphysema; the distinction between this species of prolonged expiration and that due to consolidation depends on the high pitch of the expiratory sound in cases of diffused consolidation.*

The modification in quality consists in the murmur becoming more and more concentrated and harsh, as compared with the standard. The pitch of both movements, but especially expiration, becomes higher than normal. This can be understood when we comprehend that the more dense the pulmonary tissues become, the more easily they transmit the sound occasioned by the transit of air in the bronchial tissue. We lay stress on this point, since the change in pitch is a cardinal feature of prolonged expiration associated with consolidation.*

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* Bearing in mind that auscultation is a study based on the comparative examination of the two sides of the chest, in each individual case; since the pitch of each person's respiratory murmur is a law unto itself.

Vocal Resonance and Fremitus.—The quality of vocal resonance is more concentrated than normal, and higher in pitch. The vocal fremitus is often more distinctly felt than is physiological. But in incipient phthisis the lesion is so masked by nearly normal lung tissue, that these signs may not be positive, and sometimes the other regions of the chest containing supplementally acting lung may yield results which render these methods of investigation negative.

Inspection.—As compared with the normal chest the expansion may be diminished, since the condensation of the pulmonary tissue diminishes the power of the intra-thoracic tissues to resist the extra-thoracic atmospheric pressure, but yet in some cases the tissue adjoining the area of consolidation may become vicariously emphysematous, and so maintain the contour of the thoracic walls, or even cause moderate prominence of the diseased side, or no change may be visible. It is well to view inspection as of importance to the diagnostician; but slight deviations from standards of comparison must be supported by testimony drawn from other methods of investigation.

In *using inspection as a method* of diagnosis, always fix the eyes on the same relative points on the opposite sides of the chest, for a comparison of the respiratory movements of expansion and retraction; other movements should be studied separately. If the eye is permitted to rove over the surface of the chest, there is a tendency to overlook slight differences of motion. The clavicle, or line of the ribs are the best landmarks.

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* The presence of fine crackling or other moist râles merely indicates associated bronchial catarrh, which is present in these cases, more or less, early in their history. Their localization and persistence in connection with the above physical signs, is strong corroborative evidence of incipient phthisis. (*See* chapter on râles).

Mensuration.—It is obvious that this method can only be of real service after phthisis has advanced to a material extent.

Advancing Consolidation.—It is necessary to be somewhat diagramatic in a description attempted on the basis assumed in this manual. Let us consider in the next place the analysis of a case in which diffused consolidation exists as an advance on the prior condition. There are three serious departures from the healthy standard: (*a*) The lung is more or less increasingly incapable of containing air. (*b*) The confined air is maintained in an increased state of tension. (*c*) The elasticity of the pulmonary tissue is more and more seriously impaired.

Percussion.—The resonance is always abnormally high *pitched* in these cases, since the decreased amount of air and the changed physical condition of the tissues combine to produce this result. The *quality* is subject to an interesting *variation;* it may become sub-tympanitic, or dull, according to the exact condition of the tissues. If the area over which percussion is practiced is one of consolidation, the quality will be dull; if there be much vesicular emphysema, or if the vesicular walls are largely infiltrated, and yet the vesicles are capable of being over distended with air, then, in either case, the resonance may be sub-tympanitic. By viewing the question of resonance from this standpoint, the statement can be advanced, *that in proportion to the amount of consolidation the percussion will be dull, very dull, almost flat,* *and the pitch will be high; or, the dull note will be replaced over more or less extensive areas by a tympanitic or sub-tympanitic note;* the size of the cavities containing air, the degree of intra-pulmonary tension due to loss of elasticity, together, give expression to the quality and pitch of the tympanitic resonance. It is just in these cases that the remarks on page nineteen apply, since the force and method of percussion enable one to develop very different and confusing results. To be aware of the possibility of error is, in this case, the readiest way to avoid it, but no theoretical knowledge will replace manual practice.

Recapitulation of Pathological Conditions Influencing Percussion.—The more chronic the phthisis, the more probable is it that bronchial thickening and inter-alveolar proliferation exist, but whether catarrhal pneumonia, broncho-pneumonia, or fibroid phthisis exist, the pulmonary tissues become much less elastic, the intra-pulmonary tension is proportionately increased.

The effect upon the percussion resonance has been stated. It consists in elevation of the pitch, with a dull, or tympanitic quality, and according to the amount of air contained in the underlying tissues, sometimes, a mixed type of resonance, perhaps appropriately styled tympanitic dullness, may prevail.

In addition, or in association with the preceding, small patches of vesicular emphysema may be scattered through areas of consolidation. The frequent location of this lesion is the entire anterior surface of the upper lobe, completely concealing the condition of the lobe beneath.*

The tympanitic resonance in this condition of lung

* Louis says that this condition is most common to the left base, or right apex.

may be so distinct as to generate the suspicion of formation of cavity. Auscultation must be invoked to decide the question. The difference between hollow breathing and the exaggerated or broncho-vesicular is beyond dispute. The physical signs by percussion in the *initial and terminal stages of acute pneumonia* were referred to this place. In the outset the resonance gradually increases in pitch, and loses in pulmonary quality until very marked dullness is developed. In the ultimate stage the retrogressive metamorphosis of the exudation leads the type of resonance gradually back to the normal pulmonary. In the condition known as *splenification* or *hepatization* of the lung, the resonance is also very high pitched and dull, the degree depending on the amount of exudation.*

Sources of Error.—The metamorphosis just described may be masked by the confusion of resonance rendered possible in examination, especially of the left chest, by the distention of the stomach to an unusual size. It can encroach on the thorax as high as the third rib in the axillary region. The tympanitic resonance is sometimes so low as to render percussion a negative sign. The distention of the stomach can be detected with the aid of auscultation, by causing the patient to drink a small quantity of water, which, when it drops into the stomach, will occasion a tinkle very easily detected by auscultation in the left axilla. Another source of confusion is found in those individuals in whom, from any cause, the tissue forming the chest walls becomes thin and tensely stretched over the chest; a careful method of percussion is the best resource under the circumstances. Practice light percussion (*see* page 19), so that delicate modifications of pitch may be noted. Direct percussion upon the clavicle without a pleximeter will often be a helpful method; one can make very accurate deductions as to the condition of the entire side from percussion made on this bone.*

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* *See* account of hepatization of lung; *see* Index,

Percussion of Normal Areas.—In these different *forms of consolidation* there exist always districts of pulmonary substances, more or less enlarged, which have become supplementally burdened with the respiratory function. To illustrate, when a single lobe or more upon one side is functionally disabled, the balance of the tissues substitute in carrying on respiration. The more acutely the consolidation is developed, the more markedly will the substitution be evidenced to percussion. The modifications in the tissues brought about by this vicarious action depends entirely upon the length of time the substitution is required to continue. In acute pnuemonia the opposite uninvolved pulmonary tissue is merely hyper-distended and returns to its normal textural state with convalescence from the disease: the same is true when there is a pleural effusion filling one of the pleural cavities, the uninvolved lung acts supplementally. But in chronic conditions of phthisis, or pleural lesion, atrophic intra-lobular changes result more or less completely in the development of substantive emphysema.

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* In general terms bone tissue yields a dull note on percussion. If the bone is flat, or nearly so, and encloses a considerable amount of air-containing tissue, *i. e.*, the ribs, the resonance is high pitched and the quality is subtympanitic rather than dull, as compared with the remainder of the lung. This happens because the pulses of air are shorter, since the shock of percussion is diminished by the bone. If the resonance over bone acquires a very dull or flat quality, there is probably more or less consolidation beneath. These points pertain to such bones as the ribs, clavicle, and sternum. The scapulæ are largely enveloped by muscle, so that the resonance is always dull.

The percussion resonance is the vesiculo-tympanitic, more or less high-pitched according to the loss of elasticity, and the increase of intra-pulmonary tension.

CHAPTER V.

AUSCULTATION—ADVANCING CONSOLIDATION.

In harmonizing the results of auscultation with percussion, in the examination of parallel cases by the former method, sedulously keep the fact in view that in proportion to the amount of consolidation present, the respiratory murmur advances from the harsh or broncho-vesicular to the purely bronchial. In a typical case of diffused vesicular consolidation, it is the concentration of the respiratory murmur together with the elevation of the pitch, which enables us to discriminate. Fancy vesicular consolidation so closely distributed throughout an area of lung that the respiratory murmur is restricted to the bronchial tissue; necessarily it is very concentrated, very high pitched, nearly tubular. But in catarrhal phthisis the solidified vesicles are scattered through the diseased district, areas of vesicular emphysema abound, and so the respiratory murmur is rarely tubular, but is mostly classed according to its degree of concentration and elevation of pitch, as bronchial or *broncho-vesicular*.*

In the cases of broncho-pneumonia and fibroid phthisis the respiratory murmur is also bronchial, since there is such an undue proportion of non-elastic tissue, causing induration of the bronchial and intervesicular tissues respectively. The diagnosis of these forms of inflammatory action is much assisted by inspection, and a study of the duration and etiology; for instance, the bronchial and fibroid forms of phthisis are always associated with more or less vesicular emphysema.

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* This applies to the condition of respiratory murmur during the initial and terminal stages of acute croupus pneumonia, in which congestion and partial exudation, or more or less rapid resolution are the pathological conditions.

Rhythm.—The most important observations connected with the proper appraisement of the forms of bronchial breathing are those relating to rhythm. The inherent resiliency of the lung tissue is essential to a physiological rhythmical performance of respiration. How do the alterations just advanced affect the rhythm of the respiratory act? First, the proper expansion of the lobes depends very much upon the descent of the diaphragm, and the expansion of the base of the chest. Now, the longer the lesion persists, especially if it be unilateral, the more complete is the supplemental action of the unaffected side, and thus diaphragmatic breathing is not as much exaggerated as inferior and superior costal respiration.

Secondly, the pulmonary circulation depends largely on aeration of the blood; the circulation will be most active at those sites at which the best aeration can be effected. As the result of these observations, when extensive diffused areas of consolidation exist, the respiratory murmur will be most marked upon the least affected side.

Results of a Study of Rhythm.—The above paragraph explains why we may, and often do, hear only the inspiratory portion of the respiratory movement. If there is deficient diaphragmatic movement, there is a functional tendency to distribute the bulk of the air to the most normal tissues; and the lessened demand, in many cases of chronic phthisis, upon the respiratory function favors *superficial types* of respiration over the diseased regions.

Since the lung may not be fully inflated, but a part of the respiratory murmur may be audible, and that part is inspiration, yet, if the quality is concentrated, and the pitch high, the breathing can be called bronchial. In the event of expiration being audible, it may be a mere whiff, yet, in other cases both inspiration and expiration may be audible. It is especially in the posterior portions of the lungs that the respiration is lost during the expiratory movement. In cases of atrophic emphysema interlaced with phthisis, the respiration is especially feeble. To define this modification of the respiratory murmur by describing it, we would say that *feeble respiration* implies a want of intensity in the complete respiratory act; indeed, at times the respiratory murmur seems absent.

There is one ultimate method of resolving doubts as to the pathology of these cases of feeble breathing. *Command the patient to cough* or to take a series of quick inspirations and expirations, which will enable the observer to adjudicate the pitch and quality of the respiration.*

Local Emphysema.—This is frequently the condition of large areas of lung tissue in close juxtaposition with areas of consolidation. Usually the upper lobe, especially the right, or the inferior left lobe (Louis) undergoes this alteration. Beneath, and distributed through these districts, are areas of consolidation. This is indicated by a sort of double character in the respiratory murmur, a superficial respiration, perhaps vesicular, perhaps broncho-vesicular, and the beneath murmur in quality plainly bronchial.

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* Summary of Causes of Feeble Breathing. Emphysema, from deposits in pulmonary texture, obstruction of bronchial tubes. Also feeble breathing and *varieties* of rhythm associated with pleural adhesions.

Hypostatic Congestion.—The grade of bronchial breathing is appropriate to the physical conditions (amount of condensation) present. But the diagnosis of this condition is an example of the associated study of clinical environment.

We detect hypostatic congestion during the course of zymotic disease, also in those long bed-ridden, with feeble hearts. Sometimes it occurs in the course of acute rheumatism. These points, with the bilateral distribution, separate it from acute pneumonia, from which it is also differentiated by the absence of the dyspnœa, or appropriate thermometrical record. The environment separates it from chronic phthisis.*

Auscultation of Normal Areas.—On page 64 conditions are summed up under which supplemental expiration and consequent over-distention of pulmonary substance occur. The normal respiratory murmur is exaggerated, or it is even broncho-vesicular, of typical rhythm, that is, heard on inspiration and expiration. It is sometimes so intensely loud, on account of the large amount of air passing in an out of the pulmonary substance, that it is possible to mistake the character of the murmur, and fancy that some textural alteration has occurred. To guard against this, analyze the quality of the sound, and it will be found to lack the concentration of bronchial, or broncho-vesicular breathing indicative of tissue alteration, and an analysis of the pitch will reveal that the expiration is a *low*-pitched sound, not *high*-pitched, as it would be if consolidation had occurred. These two points, the lack of concentration in the quality of the sound and the lowness of the pitch, are cardinal attributes of exaggerated respiratory murmur; the murmur may be never so loud, and yet no textural alteration is indicated, but simply an overplus of air, and consequently slight increase of tension, slight loss of pulmonary elasticity.

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* The condition itself is one of engorgement or intense congestion of pulmonary substance; it is important to distinguish it from true pneumonia, in view of prognosis and treatment. Associated with signs of auscultation, there is moderate dullness on percussion, and fine crepitant or subcrepitant râles may be heard, *bilaterally*, pneumonia being usually unilateral.

Vocal Resonance and Fremitus.—Space forbids much beyond the mention of the fundamental fact that condensation of the pulmonary parenchyma, increased tension, and loss of elasticity, all combine to favor the increased conduction of vocal resonance and fremitus; the more pronounced these departures from standard ideals, viz., condition of healthy lung, the more definite do the results of vocal resonance and fremitus become.

Exceptions.—It has been said that in the above conditions vocal resonance is heard more or less distinctly as bronchophony, but confusion sometimes arises, from the following facts. Where lesions are scattered, the intensity of vocal resonance may seem equal on both sides. It is chiefly differentiated by giving attention to the *concentration* of the sound, which, with the elevation of pitch and bronchial quality are the peculiarity of bronchophony. Exaggerated vocal resonance is merely intensely *loud vocal resonance* of normal pitch and quality. This bears out the remark that intensity does not constitute the essence of the varieties of bronchial vocal resonance. The recognition of vocal resonance sometimes extends the key to the proper appreciation of the variety of respiratory murmur present, and should be practiced if doubt exists as the quality of breathing.

Vocal Fremitus.—The best situations for the recognition of vocal fremitus are the locations at the apices and the axillary regions. The exceptions to the usefulness of the sign for diagnostic purposes are to be found in the fact that in many cases of condensation of the lungs the process, if advanced, so far precludes the admission of air into the lungs as to prevent sufficient pulsations of air to be appreciable by the tactile sense.* In addition to this, if there be much disseminated emphysema, if there be any broncho-pneumonia or fibroid phthisis, the evidence from palpation is apt to be negative.

Inspection teaches that its revelations are dependent on the physiological facts that the distention of the chest is maintained by three factors: the existence of the air within the chest, the elasticity of the lungs, and the inherent resiliency of the thoracic walls, all resisting atmospheric pressure from without. In phthisis the two first factors are withdrawn; the result reveals a deficient expansion with inspiration, and a retraction proportionate to the amount of condensation of the lung substance and dependent on the want of resistance within the chest to atmospheric pressure from without. The retraction is most apparent at the apices, since lesion is oftenest chiefly developed in them, but in addition, at the inferior portions of the chest retraction sometimes occurs, from consolidation, but *more frequently* from *pleurisy.* We understand the word phthisis includes atelectasis from the pressure of a tumor in the mediastinum, on the bronchial tubes, the occurrence of, or similar pressure from tumor of the lung (cancer or hydatid), hemorrhagic infarction, or the temporary shirking of air, owing to the filling up of the vesicle with blood.

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* The absence of vocal fremitus is a cardinal physical sign, if associated with the other physical signs of pleurisy with effusion. See Pleurisy with Effusion.

Your reason is appealed to to picture conditions sufficient to shut off the air from the chest. As instances of these rarer conditions, the writer has seen a pneumonic exudation of the upper lobe, so complete as to obliterate the bronchial tube leading to an apex, and cut off the entrance of air into the bronchial tree, so as completely to abolish the respiratory murmur and the respiratory movement. Another example is the pressure exerted on the bronchial tube by the distended aorta, in cases of aortic aneurism. Such a tumor is capable of reducing materially the respiratory movement by its pressure, either on the bronchial tubes or on the pulmonary structure itself. It has been just now said that the distention of the thorax is chiefly maintained by the within air; but there is also a thoracic resilience which assists the expansion and retraction of the thorax; it is by this prerogative that the lower portion of the chest usually presents an unaltered contour, even in advanced consolidations. Quite the opposite condition, for the most part, prevails in pleurisies of chronic type, in which pleural adhesions are almost invariable, and these exert a retractive force sufficient to overcome the eccentric resilience. This is so unvarying that, with *propriety,* a rule may be formulated endorsing the statement that *retractions* of the *apices* indicate *phthisis,* while *retractions* of one or both of the *bases* indicate *chronic pleurisy.*

Even in practicing inspection the value of the blending of clinical observation is recognized. We can by its aid differentiate cancerous tumors, hemorrhagic infarction, aneurisms, acute pneumonias, or paralysis of respiratory movement traceable to some lesion in the central nervous system, from the physical signs of phthisis by inspection.* But inspection is also helpful in enabling us, sometimes tentatively, sometimes positively, to differentiate between phases of phthisis. Let us formulate another guiding principle. The greater the amount of fibroid tissue developed in the type of phthisis, the greater the amount of external alteration of contour will result. Not only so, but displacements of the intrathoracic movable organs will occur. At the outset, let me say that, in fibroid phthisis we have an exception to the rule assigning retractions of the base of the chest to pleurisy; the contractile power of the newly developed cicatricial tissue is so great that thoracic resilience cannot contend against it, and retractions occur. To be systematic, note (*a*) broncho-pneumonia develops a tendency to those changes in the appearance of the thoracic walls typical of emphysema, elsewhere described, but without the same distention; (*b*) fibroid phthisis presents retractions already indicated, but also displacements of the heart toward the affected side, which may be so pronounced that the apex beat will be carried into one of the axillary spaces, and upward as high as the second interspace in that region. The trachea may be displaced to the one or other side, and the dip toward the abnormal side can be both felt, as well as noticed. These two displacements together form an important evidence of the development of the condition. It often happens that the processes of fibroid phthisis and broncho-pneumonia are blended. In this case, the evidence by inspection partakes of both types. The importance of the clinical study of the associated symptomatology and etiology in the diagnosis of these types of disease has already been cited. Finally, inspection leads us to notice the type of chest presented. Elsewhere due attention will be paid to the emphysematous and rachitic thorax.

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* Local gangrene on a small scale occurs sometimes around cavities in the lungs, or in the bronchial tubes, giving rise to temporary fetor of breath, but is not likely to lead to fallacious inferences, chiefly because of its temporary character.

The phthisical chest has been represented as long, flat, coffin-shaped. The shoulders stoop, the spine is carried forward, the ribs are more oblique, depressing the sternum, elevating and tilting outward the scapulæ, the result giving the alar or winged appearance. There is this much to be said relative to the share of this contour of the chest in the development of phthisis. The chest capacity is hence reduced by the awkward position of the muscles, and when catarrhs develop, the disposition to become chronic is very much enhanced. The importance of this shaped chest as a predisposing factor to phthisis has been exaggerated.

Exceptional Phenomena.—When there is pressure on the bronchial or laryngeal tissue, the expansion of the lung does not follow the expansion of the chest walls, which are carried outward by their inherent *elasticity,* so that the intercostal tissues are depressed.

There is another condition under which the intercostal tissues move *inward on inspiration*, viz., in many cases of pleurisy, with and without effusion, as will be explained in section on Pleural Alterations. This is quite apart from the condition of retraction in chronic pleurisies with adhesions, for this occurs notably with the *expiration.*

Mensuration is in most respects the complement of inspection; the value of tracing a plan of the chest on paper (*see* introductory remarks on mensuration), is to make a record for references. By this means the contour of the chest is presented to the eye. Circumferential measurements are not to be relied on, save in cases of extreme retraction; for further comments on this subject consult section on unilateral enlargement, under title of Emphysema.

CHAPTER VI.

INDEPENDENT MODIFICATIONS OF RESPIRATORY RHYTHM.

Apart from the conditions of consolidation considered as involving the various tissues of the pulmonary substance, there are certain departures from typical standards which can only be understood and described by themselves.

The most important method of diagnosis of these departures is auscultation. The attribute of respiratory murmur which is modified is rhythm. We have already spoken of modifications of rhythm in early phthisis, and shall allude subsequently to its important modification in diseases of the pleural membranes. At present we divide the pathological conditions influencing rhythm into two classes. (*a*) Modifications of respiratory rhythm synchronous with cardiac rhythm. (*b*) Modifications simply of respiratory rhythm. (*c*) A sub-classification into permanent and temporary causes. The first class, (*a*) also permanent, includes pressure of a large heart on the bronchial tubes, pressure of an aneurism upon the lungs, or bronchial tubes; pressure of moderate solidification of lungs on the subclavian artery. This is a condition associated with a murmur in the artery, to be spoken of subsequently, in an analysis of subclavian murmur. In addition, pleuro-pericardial adhesions will *often pro*duce a noticeable interruption of rhythm. In class (*b*) the pressure of a tumor in mediastinum on the bronchial tubes, tumors of the lung—"hydatid," "specific," cancerous—are all most liable to influence the rhythm during their persistence. In class (*c*) we find some temporary conditions which are apropos. Among these belong collections of inflammatory or other fluids, such as blood or the serum of œdema.

The *quality and pitch* of the respiratory murmur is influenced by the principles previously laid down, and the modifications of the vesicular murmur will accord with the physical condition of the tissues in point of density, elasticity and tension. One point is very noticeable, viz., that whatever be the quality or pitch of the respiratory murmur, it is often very feeble. In cases of pressure on the bronchial tree, or mediastinum, the bronchial breathing may be high pitched and whistling in quality, in proportion to the lessening of its calibre, or, on the other hand, a bronchial tube may be compressed, and its lumen positively obliterated. Caution must be exercised in the latter case, not to mistake the loud murmur in an opposite bronchial tube to be pathological, a mistake easily made since the tissues concerned are deeply situated.

The spot at which to auscult for evidence of pressure on the bronchial tubes is over the second dorsal vertebra, but all the other methods of physical diagnosis may be invoked, and conducted under general principles. As additional instances in this study of the modifications of rhythm, we observe the *neuralgias* of the intercostal nerves or muscles, and angina pectoris.

To make a differential diagnosis between the intercostal neuralgias and pleural or pulmonary disease, observe two facts: the neuralgias are associated not only with pain, but also with *tenderness* on pressure, the pulmonary processes by pain alone.

The second fact is that neuralgias are unattended by fever; the reverse prevails in the opposite conditions. For the diagnosis of angina pectoris we refer to its appropriate section.

Conditions within the abdominal cavity, dropsies, tympanites, enlargement of the viscera, morbid growths, are capable of seriously modifying the pitch, the quality and the rhythm of the aspiratory murmur. The further discussion will be found under section upon abdominal conditions, and their influence upon thoracic examinations.

CHAPTER VII.

ADVENTITIOUS CAVITIES IN THE PULMONARY SUBSTANCE.

Auscultation and percussion are the most available methods of diagnosis.

Percussion demands that the cavity should be near the surface; auscultation requires that the cavity shall be at least as large as a walnut. Let us first study the evidence pertinent to the pathological formation of cavities, together with their anatomical relations and environments, finally bringing to our aid a knowledge of the general symptomatology, and then the physical diagnosis can be discussed.

The formation of cavities in the lungs is a sequence in the course of the various forms of phthisis. Subacute inflammations, pneumonia, and gangrene are all elements in the development of cavities. The process by which these losses of substance occur may be briefly collaborated—(*a*) by a slow or rapid process of fatty degeneration, followed by ulceration; (*b*) as the result of chronic bronchitis, and softening of bronchial tissue, with subsequent yielding to traction from without; for instance, in broncho-pneumonia or fibroid phthisis; (*c*) abscesses as a sequence of acute lobar pneumonia, following hepatization or purulent infiltration; (*d*) as the direct result of gangrene, itself the immediate consequence of wounds of the lung, or blood poisoning, or of emboli.*

It naturally follows that there are two locations for vomicæ, viz., the pulmonary and the bronchial tissue. In the pulmonary substance they are situated most frequently at the apices, but may occur at the base of the lungs. They are usually small, though they vary in size from a walnut to an orange, or even larger, or there may be multiple small sacs opening into a bronchial tube, like so many blind pouches. In the latter case, the appearance of a section of the lung resembles a large honeycomb. A single cavity may also terminate in a bronchial tube. Cavities are situated more or less superficially. The walls of the deeper cavities are composed of solidified pulmonary tissue, or they may be made up of patches of uninvolved tissue, or tissue in a state of vesicular emphysema. It is this fact which renders auscultation a valuable means of recognizing deeply situated cavities which would be passed over unnoticed if we relied only upon percussion. If we observe the quality of voice and respiratory murmur, we cannot be misled.

There is one especially notable fact in the environment of large superficial cavities, viz., that they are often (but not always) attached to the chest walls by adhesive inflammation between the visceral and costal pleura. This inflammation is developed in the pleura by contiguity of inflammation in the lung. The presence or absence of this adhesion of the pleura is a factor of great importance in physical diagnosis, since it prevents the pulmonary tissue from collapsing on expiration, thus maintaining the walls of the cavity in a state of more or less distention. Of course there must be many different degrees of thickness of cavity walls, in these more superficial vomicæ. Their walls may be so thin as to be composed in many cases of the pulmonary pleura only. But the adhesion of the cavity to the costal pleura has another significance. It prevents the development of pneumothorax through rupture of the cavity wall, a probable accident when the cavity wall becomes so thin. (*See* Pneumothorax.) The mere situation of a cavity of this sort is of aid to diagnosis. Cavities due to *pulmonary abscess* are situated usually (although there are exceptions to this rule) in the base of one lung, the other lung being usually healthy. Phthisical cavities are located in one or both upper lobes, and if in one lung alone, the same lung is more or less infiltrated, but both sides are usually involved, although lesions are commonly furthest advanced in one of the lungs. (*b*) In the division including cavities in the bronchial tissue, dilatation takes place in two forms: either the tubes are uniformly dilated like the fingers of a glove, or else they form cavities by undergoing saccular enlargement. The cavities are usually situated at the middle or lower third of the chest, and may be strung in a row following the distribution of the bronchial tubes. It consequently follows that these cavities are recognized anteriorly as located near the second and third costo-sternal junction, or posteriorly somewhere in the near vicinity of the third dorsal vertebra, at which point the bronchial tubes are superficially situated. The cavities may be unilateral or bilateral, according as they accompany chronic bronchitis or fibroid phthisis. The percussion resonance is not commonly much impaired, not nearly so profoundly as it is around a phthisical cavity.

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* Local gangrene, on a small scale, occurs sometimes around cavities in the lungs, or in the bronchial tubes, may give rise to *temporary fœtor of breath*, but is not likely to lead to fallacious inferences, chiefly because of its temporary character and the absence of permanent concomitant symptoms.

Clinical Evidence.—The associated clinical evidence is manifestly a most important aid in correctly interpreting the results of phthisical diagnosis.

Bronchial Dilatation.—Frequently for years the general health is almost unimpaired, and it is never impaired proportionately to the degree indicated by the physical signs. There is no hemorrhage or night sweats, and emaciation is not a pronounced symptom. The same physical signs persist for months or years unchanged, contrary to the history of most phthisical cavities, which continually alter with the advancing malady. The expectoration of bronchial dilatation is more abundant and fluid than in catarrhal phthisis, it is more copious and purulent (*see* Fibroid Phthisis), and is usually brought up in morning or evening by the cupful; it is not a constant spitting of nummular sputum, as in true consumption. In chronic cases the expectoration may become so fetid as to generate suspicions of gangrene; the cough is harrassing, but is often relieved if the bronchial cavity is thoroughly emptied. These particulars are given because these cases are often mistaken for true consumption, whereas their prognosis is very much more favorable, though it is not within the scope of this handbook to treat thereon.

Cavities of the Nature of Abscesses.—The pathology of these cavities, with its coincident clinical history, is not that of phthisis. The history of these cavities is either recovery by contraction (especially after wounds), or more frequently the abscess grows larger and larger till the entire lung may be destroyed, in this respect resembling phthisical cavities. When death occurs it is by exhaustion and hectic; where recovery takes place it is by free opening externally, or internally, and evacuation of the contents. At times the small amount of constitutional disturbance, slight degree of emaciation, the comparatively good pulse, easy breathing, slight cough, and comparatively healthy complexion are in noticeable contrast with the physical signs. Cavities of the nature just described are mostly located in the base of the lungs.

Phthisical Cavities commonly are situated in one or both lungs, and are indicated as a development in a train of symptoms which include as prominent features gradual emaciations, persistent loss of weight, by reason of mal-assimilation of food, more or less frequent hemorrhage, more or less hectic, frequent pulse, hacking intermittent cough, nummular sputum, expectorated in varying amounts throughout the twenty-four hours,* and preëminently remarkable from their slow development, and genesis in association with the gradual development of some of the tissue modifications of one of the recognized forms of consumption.

We are now prepared to analyze the physical diagnosis of cavities :—

Percussion.—In order to appreciate the percussion note of cavities we must have in our own mind some standard of comparison ; this standard is furnished by the percussion note of the small intestine. The reverberation will then, primarily considered, be denominated of tympanitic (or empty) quality, of high pitch, as compared with other pulmonary percussion resonance, or with the ideal tympanitic note of the stomach. The physical conditions are an empty space or spaces in the lungs or bronchial tissue, at varying depths from the surface of the chest, and surrounded by walls of varying conditions of tension and elasticity.

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* Not periodically as in bronchial dilatations, nor inaugurated by a gush of pus, and mucus as in abscess.

Modifications.—First, if the cavity is deeply situated, the sound may be simply a tubular note without hollowness, in which case the cavity can be diagnosticated only by auscultation.

On the other hand, if the cavity be very superficial, if it consequently possesses thin walls, those frequently adherent to the costal pleura, the hollowness will become most pronounced, the pitch very high, and the expression amphoric tympany is used. Amphoric is a word taken from amphoræ, a jar, and represents to us the resonance of a thin glass vase, which is a very hollow, high-pitched sound. The cavity yielding this sound most typically is one with walls rendered tense by adhesions. Between the tympanitic as just differentiated from the tubular, and the amphoric, every gradation of pitch will be found, but all these are included under the term hollow, or cavernous. The sounds are dependent for their pitch on the proximity or remoteness of the cavity from the surface, and upon the thickness or thinness of the chest walls.

If a cavity is located at the apex of the lung, if the chest walls become very thin, the abnormal resonance may be very pronounced.

Cracked-Pot Resonance, is a term adjectively used to describe a jingle, or broken quality grafted into the hollow resonance indicative of cavity. The sound may be imitated by striking the clasped hands on the knee, as is sometimes practiced to amuse infants; sometimes the sound is as broken as the resonance of a cracked goblet. Two sorts of cavities yield this percussion, the single superficial cavity communicating with a bronchial tube, or a series of small cavities terminating in one opening leading into a bronchial tube. In this latter case, the illustration just furnished gives the idea of an explanation of the resonance, for just as the air is expressed through the fingers when struck against the knees, so the air is driven by the shock of percussion out of the cavities, or loculi, and into a bronchial tube. One of the striking peculiarities of the sound is, that the cracked-pot quality is a transient attribute, in many cases, of this phase of tympanitic resonance. The jingle has seemed to the writer due to the transit of the air through the mucus (used generically for fluid) in the bronchial tubes entering the cavity. Coughing or expectoration will often abrogate the quality, which half an hour later may return. It is possible that the sound may be developed by some conditions of cavity wall not already noted. However this may be, the essential point is that there must be an open bronchial tube leading into a cavity.

"*A cracked-pot sound* is possible as a glottidean sound (Wintrich) produced by the rapid irregular vibration of the vocal cords." The explanation refers to the species of cracked-pot sound in cases of children, in whom the chest walls are thin and yielding, and the air can be readily expelled from the bronchial tubes by percussion.

In a few adults with healthy lungs, but thin, yielding chests, a cracked-pot sound is possible.

Methods.—There is a peculiarity of percussion stroke designed to give the most favorable expression to this species of resonance. The blow should be heavier than usual, the plexor being allowed to remain an instant on the finger used as a pleximeter. This implies, therefore, a more decidedly deliberate percussion than is usually advised. It is also well to cause the patient to rest the back against the wall, or a door, before percussion is attempted. Finally, direct the patient to open the mouth, the ear of the listener being placed as close to the mouth as possible (especially if the sound is not immediately distinct, when the mouth is closed), for by this means the air tubes act as telephones, transmitting the most feeble cracked-pot quality to the ear. *Value.* In our opinion the sound is a most valuable indication of cavity, since the exceptional cases can so easily be differentiated.

Auscultation.—The variation from the tubular type consists essentially in the change from the concentrated high-pitched sound, to some tone of hollowness.

The word cavernous is, qualitatively speaking, universally expressive of the respiratory murmur; in these cases the idea of a thick or thin-walled, empty space, can also be obtained, hence the word amphoric respiration is sometimes used, which simply means superficial emptiness. The tone is also to be studied, and we have high and low-pitched hollow breathing. Any deeply situated cavity, whether separated from the ear by its depth within the pulmonary substance or beneath the tissues covering the chest, will give a cavernous or *muffled hollowness*, usually of low pitch, as compared with tubular breathing. The reason for this is that, since the cavity is deeply situated, its walls can expand on inspiration and collapse on expiration, instead of being unyielding, as the bronchial tissue. On the contrary, the superficial cavities usually have tense walls; these do not collapse on expiration; thus the echo is more high-pitched, and the hollowness is termed amphoric, or high-pitched empty sound. Now, between the two extremes any gradation can occur.

The amphoric respiration grades into cavernous, and it in turn grades into tubular respiration.

Modifications of Respiratory Murmur Due to Situation of Cavities.—Cavities situated near the apex anteriorly will, according to the preceding principle, yield an amphoric respiratory murmur of typical timbre—and the same-sized cavity situated in the scapular region posteriorly, may from the density and thickness of the external tissues at that situation yield a cavernous breathing. If the rhythm of the respiratory murmur be normally maintained, these sounds are typically developed. But feeble inspiration may mask them completely. It is of utmost importance that full inflation of the chest shall occur. This is to be secured sometimes by causing the patient to cough, and sometimes by desiring a series of quick but short inspirations.

Auscultation of Voice.—One most successful part of the examination by auscultation results from a study of the vocal resonance. Aside from the quality of voice, the attempt will frequently shed light upon the appreciation of the respiratory murmur. The quality of hollowness of voice is remarkable: the phonation is caught up and repeated until it seems as though the sound must be developed immediately beneath the ear; to this resonance the term pectoriloquy is applied.

Pectoriloquy is developed by two species of phonation, audible and whispered. The first is often too reverberant and intense to be discriminated from bronchophony, or exaggerated voice, but *whispered* pectoriloquy is very diagnostic.

Modifications.—The vocal resonance is a most valuble aid in the recognition of deeply seated cavities, for instance those developed in the scapular region posteriorly.

The only source of error is to mistake slightly enlarged or really dilated bronchial tubes for *cavities.* If regard is paid to location, and the symptomatological environment of bronchial dilatation, these errors can be avoided.

The results of a study of vocal fremitus are negative, since the air within a cavity diffuses the waves of the voice so that they cannot be recognized by the sense of touch.

Inspection can at most show a pronounced retraction of the chest walls; palpation is negative.

CHAPTER VIII.

EMPHYSEMA.

The tenor of our remarks has been designed to encourage reflection upon the effects of departures from the physiological pulmonary condition occasioned by widely different causes. But there are certain conditions to which special reference must be made; one of these seems in place at this point in our study.

Pathological Anatomy and Etiology of Emphysema.—The morbid physiological process of general pulmonary emphysema consists in a progressive dilatation of the air sacs, with more or less destruction of their septa, and a consequent associated increase in the bulk of the lung hypertrophy, and in this respect it differs from vicarious emphysema, and receives the designation substantive.

The atrophy of the septæ and dilatation of the air vesicles results in an obliteration of the capillaries, a limitation of the aerating surface for their ramifications, consequently there is an interference with the flow of blood through the lungs, and the many consequences of venous repletion ensue. From an etiological standpoint emphysema is frequently the sequel of chronic bronchitis, it is also a sequel of recurrent attacks of asthma, and it is developed in those who have played for years on wind instruments.

These various causes result, in one way or another, in the gradual increase of the residual air in the lungs, from the difficulty in performing expiration. It is essentially a chronic malady, but in its course subacute attacks of bronchitis occur, which much augment the difficulty of respiration. The bronchitis results in the greater or less blocking of the bronchial tubes with cells and mucus. The air is drawn into the chest by the power of the inspiratory muscles and the pulmonary elasticity, but the air cannot so easily pass out again. It is this which in the course of the disease so much annoys the patient; his whole effort is to force the air out of the pulmonary tissue. The effects of emphysema are manifested in twofold guise, the local tissue changes and in the general symptomatology.

The obliteration of the blood vessels results in the sense of constriction in the chest, the dyspnœa, the anxious look, the bluish lip, characteristic of emphysematous patients, and consequent upon this results the dilation or dilated hypertrophy of the right side of the heart.

The manifold effects of deficient oxidation are too numerous to mention here, but prominent is the lesion of fatty degeneration of the tissues. The imperfect circulation in the chylopoetic system results in imperfect assimilation, indigestion, tympanites, and all along the lapse of years before the patient succumbs is a liability to repeated attacks of bronchitis from the continued repletion of the radicles of the pulmonary artery. In the end transient dropsies may indicate the overloaded state of the right heart. The appreciation of the local tissue changes is the aim of the physical diagnostician; let us at once proceed to this study. The physical signs are bilateral, unless there are chronic lesions in one lung such as to forbid its expression. When, for instance, one lung is cirrhosed, or greatly collapsed from unyielding pleural adhesions, it is common to find emphysema of the other lung.* The increase in bulk of the lung results in a displacement of the liver downward to a variable distance; the heart is displaced downward, and the enlargement of the right ventricle increases the size of the area of cardiac dullness, to the right of the sternum, evidenced by pulsation of the right ventricle in the epigastrium; of less importance we have displacements of the stomach and spleen. These latter facts are interesting in view of the tympanites and indigestion of emphysematous patients. Finally the increase in bulk of lung results in the bulging of the lungs above the clavicles, especially during coughing or powerful expiration, and the *heart and liver are enveloped* so that their outline can scarely be defined by percussion. *Percussion* results in a widespread tympany, of a pitch which is variable, dependent, of course, on the degree of emphysema, and the consequent loss of pulmonary elasticity and increase of pulmonary tension. It is always a *lower-pitched note* than can be developed over pulmonary cavities, since the tension is never so great, nor is the elasticity as completely destroyed. It is a higher pitched tympany than the stomach, since the size of the cavities containing the air are smaller, and the vibration of the pulses of air is necessarily shorter. The normal areas of liver and cardiac dullness are obliterated, and in place only tympany will be developed, but the tympany will be often more high pitched at these situations.

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* In chronic pulmonary diseases, Louis tells us the upper lobe of the right lung, or the lower lobe of the left are the parts most frequently emphysematous, and present visible local bulging

Auscultation.—The quality of the respiratory murmur is variable, proportionate to the amount of hypertrophied tissue. If the emphysema is general and pronounced, the respiratory murmur is lost. Since the vesicular structure is permanently distended with residual air, we do not hear the vesicular part of inspiration due to gradual vesicular expansion as in health. We do not hear vesicular collapse in physiological states; in this lesion there is no vesicular collapse, and since the bronchial tissue is muffled, the respiratory murmur is extremely feeble. The chief alteration then is in *rhythm;* the inspiration being audible as a short sound, in reality it is prolonged, the expiration notably prolonged, possibly somewhat interrupted. The most important fact to remember relates to the pitch of expiration which is low, while if there is consolidation the pitch is high. Frequently, however, the amount of vesicular emphysema is proportionately small, while the antecedent bronchitis has wrought more or less extensive bronchial alteration, dilatation and thickening; the evidence by auscultation reveals in these cases much more of a broncho-vesicular or harsh respiratory murmur than previously described. Cases of emphysema are frequently associated with râles, more or less numerous, from inter-current bronchitis, to which allusion will be made subsequently.*

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* Emphysemas preceded by bronchitis are often comprehended in the group of broncho-pneumonias, but in true emphysema there is rarely a tendency to chronic consolidation, although death may occur from pneumonia.

Vocal resonance and fremitus are negative, the diffusion of the voice yields a distant vocal resonance of normal quality. In the cases in which there has been prior bronchitis, the vocal resonance is harsh. Vocal fremitus is naturally much diminished, nearly as much as in pleural effusion, but the associated physical signs separate the condition very widely from such diseases.

Inspection and Mensuration.—By these combined methods we study *three* modifications which belong to the natural history of emphysema. 1st. The bilateral enlargement of the chest of general substantive emphysema. 2d. The unilateral enlargement due to supplemental emphysema. 3d. The pigeon chest modification of rachitis. In the first condition of general pulmonary emphysema, the modification is so marked that the impression of the tendency toward dilatation is obvious.

Speaking of typical cases, full inspiration after overcoming the obstructions inflates the chest to its utmost capacity. This of course elevates the ribs, and causes a transition of the elliptical into the circular form of chest. The antero-posterior diameter of the chest is notably increased, the vertical diameter is lessened. In a well-marked case of emphysema the thorax is in a state of distention beyond that which would have been produced during health by the deepest possible inspiration. Then comes expiration, associated with powerful diaphragmatic contraction; the lower ribs, which are free to move, are drawn inward to such a degree that there is a distinct sulcus furrowing each side of the chest, running obliquely forward; or the entire inferior portions of both sides of the chest are drawn inward, so that the upper portion having become circular, the whole chest assumes an appearance not unlike a barrel. But still further, the upper part of the sternum being less free to move than the inferior part, there is a species of arching forward of the sternum, and a sinking inward of the lower portions of the bone. To give fullest scope to inspiration, the spine arches forward, sometimes so as to render the sternum nearly vertical, and the front of the chest appears flat; the shoulders follow the spine, and thus a stooping posture is encourged. The resiliency of the chest in marked cases is so nearly nil the breathing is thrown on the upper part of the lung, and respiration becomes superior thoracic, but on the whole, the breathing in a typical case is so feeble that the chest walls scarcely move. Of course, there may be at times some diaphragmatic movement discoverable. In such cases the sternum and ribs which have become united by ossification of cartilages (see works on Practice) move up and down as one piece during the expansion of inspiration, while the inferior ribs are drawn inward during the descent of the diaphragm, to expand again on expiration. There is also a noticeable movement of the intercostal spaces. In extreme cases, as I have said, respiration is so feeble that the interspaces are on a level during the period of pause, and the pulmonary elasticity is abrogated. It is in these cases only that the tissues sometimes *bulge on inspiration.* In less marked cases the eccentric thoracic resilience is opposed by some remaining *pulmonary elasticity*, and while the *chest walls are carried outward, the intercostal tissues are drawn inward during inspiration, and bulge outward during expiration.**

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* Intercostal tissues move inward. *See* also pleural effusions by inspection—Pressure on bronchial tubes; also obstruction of bronchial tubes, as in capillary bronchitis, with atelectasis in children. *See* article—In a few cases of chronic fibroid phthisis.

Once again, we cannot discover by inspection the apex beat in its physiological position. On the contrary, the impulse of the heart is observed in or near the epigastrium; it is not an apex beat, but is the pulsation caused by the thud of the enlarged right ventricle.*

A description of the greater includes the less; mark well the departures from a healthy standard toward the emphysematous type of chest. It will, in cases of phthisis, furnish the key to the estimation of the influence of bronchitis in its parentage, and give valuable aid to the therapeusis.

2. **Unilateral Enlargement.**—Its etiology is much the same as bilateral enlargement, though it is not so constantly substantive; its causes are those which demand supplemental activity of the lung; for instance, disablement of opposite lung, pleural effusion, etc.

The only point worthy of attention pertains to mensuration. Circumferential measurements of the one or both sides are obviously capable of yielding results; but when one side is to be compared with the other by measurement, remember "that considerable increase in the sectional area of the chest may occur, and the length of the periphery remain the same by the passage of the elliptical form into the circular, and next, that the displacement of the mediastinum, which accompanies unilateral enlargement, thrusts the heart into the unaffected side. Add this consideration too, that the walls of the healthy side must follow the antero-posterior projection of the diseased side, and then it will be plain why, as a matter of fact, the perimeter of the expanded side often measures very little more, nay even less, than that of the side which is not diseased."* A tracing on paper, by indicating not merely circumference, but also shape, affords us the true means of recording the amount of unilateral enlargement. 3. The pigeon and rachitic change referred to in the section on inspection of the normal chest, is often associated with more or less emphysema, usually consequent on the bronchitis, due to the effects of imperfect pulmonary expansion, to which the deformity renders them liable.

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* Causes of epigastric pulsation in order of frequency:—

1st. Enlarged right ventricle.

2d. Pulsation of abdominal aneurism.

3d. Aortic pulsation communicated to tumors in the abdominal cavity.

4th. Enlarged and pulsating liver, in connection with disease of mitral or tricuspid valves.

5th. Tremor of normal tissues transmitted from the aorta, if there be some tympanitic distention of abdominal viscera.

6th. Displaced left ventricle in cases of acute or chronic pleurisy.

Inspection at once recognizes these deformities, and directs attention to incident pathological changes, which in turn reflect light upon the results of auscultation and percussion.

Diagnosis.—In view of what has been said, the diagnosis is not likely to occasion difficulty. The principle conditions resulting in considerable thoracic enlargement unilaterally, viz., pleural effusion, or rapidly developing morbid growths in the lungs, or the pleura, are simply differentiated by percussion.

Pneumothorax is probably the condition most likely to be confounded with it. (*See* Pneumothorax). Bilateral enlargement from this cause would be nearly incompatible with life.

Atrophic Emphysema.—Allied to the preceding group is another form of emphysema, in which that pathological state is ultimately linked with atrophy of all the pulmonary tissues, and with atrophy and fatty degeneration, not only in the lungs, but as a widespread degenerative change, including especially fatty degeneration of the heart muscle. We find in these cases, atheroma of the arteries, arcus senilis, defective nutrition, loss of appetite, with deranged functional activity of the liver, loss of sleep, dyspnœa, all developed largely in consequence of a fatty degeneration of the heart,* and ultimately associated with its symptomatology. We have cough and evidences of bronchitis dependent on the enfeebled circulatory power, and sometimes areas of positive congestion. The tout ensemble is almost exclusively seen in middle or advanced life, but sometimes these changes occur in those in whom alcoholism, syphilis, etc., have engendered in early life these degenerations, commonly the dealings of the hand of time. It also occurs not rarely as a consequence of the arrest of previous pulmonary phthisis, and as such has been alluded to in our accounts of the tissue changes of that disease, and it is sketched as an independent change under section (*c*) page fifty-four. The pathological condition being understood the physical signs by auscultation and percussion may be found under the grouping of subacute or chronic pulmonary tissue changes in advancing phthisis. We have here to note that it occurs as a bilateral, as well as a unilateral process, and that inspection is very helpful in diagnosis. Inspection usually includes: 1st. A universally flattened chest anteriorly. 2d. It has all the fixed inexpansible look of emphysema with the changes in costal cartilages. The sterno-mastoid muscles stand out strongly against the sunken supra-clavicular spaces, unless they are bulged by a forcible expiration. 3d. The diaphragm is depressed; there is epigastric pulsation. 4th. The heart is covered by lung, as in hypertrophic emphysema. Finally, we must arrive at an absolute diagnosis by exclusion.

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* Dr. Gée.

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* *See* Symptoms of Fatty Heart.

CHAPTER IX.

RALES.

Fundamentally considered, râles are adventitious or new sounds, which are developed in the pulmonary tissues in one of four ways, and can be classified into two groups.

Group. I. Râles.

1st. By the transit of air through fluid in the bronchial tissues, or pathological excavations in the pulmonary tissue.

2d. By the transit of air through bronchial tissue, in which the natural calibre of the tubes is diminished.

3d. By the entrance of air into the vesicles. (Infrequent.)

Group II. Frictions. Sometimes called pleural râles.

4th. By the alterations of the surfaces of tissues which come in occasional contact, normally smooth, but which are pathologically roughened. The apparent quality of the sounds is the single basis of the classification. In both groups the sounds may be defined as cracklings or râles.

In group second the râles possess a distinct, rubbing quality, so that they are termed frictions. In both groups the sounds are designated by the terms dry and moist râles or friction sounds, because of the impression they make on the ear. All moist rales developed in the bronchial tissues are also described by the adjectives large, small, and intermediate-sized râles.

Another cardinal condition for the development of râles is, that the more perfectly respiration is carried on, the more satisfactorily clear will the sounds become.

It is a common observation, that on quiet, *i.e.* (often feeble), respiration, no adventitious sound can be heard, but as soon as deep respiration is attempted, at once the râles become plainly audible.

Let us take up the first class, moist râles developed in the bronchial tissue. It is plain that the generic term fluid may stand for mucus, for pus, for blood, or any other liquid; at the same time the quality of the liquid has to do with the size of the râle, which is smaller, the more tenacious the fluid. The size of the râle depends on the location in the bronchial tree at which it is generated; the larger the tube the larger the râle; when there are cavities developed in the pulmonary substance, the transit of the air through the fluid therein creates a large moist sound, compared rationally to a bubble, or jingle, which may have an amphoric echo. It is precisely upon this simple plan that the nomenclature of moist râles has been more or less minutely attempted. The sequel of efforts at inclusive designation has led to much complication. Regard all nomenclature as tentative; christen the râles on the basis just stated; using the word mucous as a generic term sanctioned and defined by precedent as inclusive of the many varieties of liquid occasioning râles.

Simply for convenience of description we would suggest the following classification:—

Mucous râles, large
“ “ bubbling } or gurgling, may have amphoric echo.
“ “ small.
“ “ very small.
“ “ crackling or subcrepitant.
“ “ *crepitant.*

Amphoric râles are to be classed as indicative of large air-containing cavities, possessing thin walls containing fluid, connected with an air tube. A metallic tinkle is a bell-like mucous sound, which can be artificially developed by striking sharply with a pin a metallic glass or porcelain cup. It is a sound developed by the splashing of particles of fluid in a cavity which possesses thin walls and is freely supplied by air. It can also arise from the splash of mucus falling into a cavity.

Tense walls pertain to conditions which are found in pneumothorax, when the opening into the lung is patulous or in a very large cavity with thin walls at the top of the lung. It is possible, but extremely unlikely, to hear a metallic note imparted to a large mucous râle developed in the bronchial tubes.

The second class are essentially dry râles, they are called

Sibilant or high-pitched hissing râles,
Sonorous or low-pitched snoring râles,

according to the size of the air-passage in the bronchial tubes in which they are generated. These râles are developed by the transit of air through the bronchial tubes, which are narrowed by a swelling of their lining mucous membrane, or by a large plug of tenacious mucus. This explains the fact that they are sometimes displaced by coughing. The sibilant râles have all grades of quality; whistling, cooing, hissing, piping, sawing, fluttering, are all terms used for their designation, according to the fancy and verbosity of the observer.

Class three. These râles are vesicular, caused by the severing of the walls of the vesicles rendered adherent by small amounts of adhesive fluid. They are called crepitant râles, a sound which resembles the clicking caused by the lighting of a chandelier by electricity. It can be imitated by the rustling of the finest silk, or the rubbing of the hair between the fingers, close to the ear, or by separating the thumb and finger rendered adherent by mucilage. The sounds are both dry and moist. The dry crepitant râle is almost limited to the incipient stage of croupous pneumonia, when exudation is just beginning to be poured out into the vesicles. It is characteristic of this râle to be developed all over the site of inflammation, and it will be remembered that an entire lobe, or at least a large section, is evaded in this disease.

The moist crepitant râle is sometimes heard in conditions of œdema, at its very outset, but the sound is bilateral and soon becomes a subcrepitant râle. Moreover, it is associated with the general conditions predisposing to œdema of the lungs. The friction râle is dry or moist, according to the stage of the pleurisy in which it is developed.

The dry friction râle is in quality a rubbing sound. It has been compared to the creaking of a new leather shoe; it is nearly always a grazing, or tearing, or grating sound. The moist friction is developed when serous surfaces, whether pleural or pericardial, are approximated, and rub against one another, especially in the early stages of inflammation. It is a very fine cracking sound, often *acoustically* indistinguishable from the fine, moist, subcrepitant râle within the lung. These râles manifestly occur both with respiratory and cardiac rhythm. They must also be differentiated from intra-pulmonary râles. Let us elaborate these points: 1st. *With reference to their differentiation from intra-pulmonary râles.*

Dry râles frequently, and moist râles occasionally, give rise to a fremitus which is not only audible, but which can be felt. In some cases of chronic bronchitis, moist râles give rise to a fremitus, but the many marked evidences of bronchitis prevents any confusion.

The true frictions are pre-eminently superficial sounds, and the respiratory murmur, with or without râles, can, with practice, be determined to be a beneath sound, developed in the deeper tissues. They are essentially grazing sounds, that is, of come-and-go quality, not commencing with inspiration but toward its close, and ending before complete expiration. The rubbing, or harsh quality of the creaking or grating varieties of friction are easily recognizable by the above means alone; not so the finer forms. In *addition*, therefore, observe, they are not influenced by the succussion of the act of coughing.

They are developed usually over the inferior portions of the chest, anteriorly or posteriorly, and frequently over a limited space. They can often be abolished by the following manœuvre, first suggested independently by Dr. Van Valzah, and the writer. When the chest is fixed, especially at the lower two-thirds, by the embrace of an assistant, or even by tightly drawn adhesive plaster, and the ear or the stethoscope is applied over the doubtful sounds they will be found to have disappeared, if of pleural origin, but to be still discernible if bronchial râles. Further, if possible, cause the patient to incline to the opposite side to that diseased, and place the hand of the diseased side, on the head, this puts the pleura in a state of tension, and often will obliterate a friction râle.

To differentiate pleural from intra-pulmonary râles is sometimes impossible, especially with the finer forms. To recapitulate the methods most approved to distinguish points of difference, 1st, the fixation of chest as described. 2d. The influence of coughing. This will disperse or alter the size and location of intra-pulmonary râles, but a friction sound will be unchanged.*

On the other hand, the grazing quality of the pleural friction, and its association with but a portion of either respiratory movement, even on full respiration, will in most cases enable us to discriminate. 3d. The ear must be trained to observe sounds generated on different planes of tissue. 4th. The association of the other means of diagnosis of acute or chronic pleurisy, or pericardial diseases must be invoked. 5th. Especially note the associations of these râles with limitation of expansion observed by inspection in acute pleurisy, or the retractions observed in chronic pleurisy. 6th. The etiology of pleurisies or pericarditis must be studied. Diagnosis between friction râles of *respiratory* and *cardiac rhythm* is considered under the section on diseases of pleura and pericardium.† Having defined the attributes of râles, it is next in place to study the conditions under which they are developed. In all the varieties of subacute or chronic inflammations of the lungs, râles are developed as the result of the softening of the inflammatory new formations, or as the result of intercurrent bronchitis, acute or chronic. Our study includes the moist râles in first and second classes of the first group. Acoustically it is impossible at times to decide whether the râles are the harbingers of the softening of the results of inflammatory action, or are the expression of an intercurrent bronchitis. In making differential inferences observe some of the following propositions: (*a*) If the râles are harbingers of softening of the lung tissue, ere long cavities with gurgling râles, and appropriate auscultation and percussion, will attest their etiology. (*b*) Moreover, they will be limited to the areas already designated by auscultation and percussion, as the areas of inflammatory new formation. (*c*) The higher range of the thermometrical record indicates the softening of the inflammatory new formation, rather than bronchitis. Sibilant and sonorous râles are frequently heard in the course of phthisis, indicating either an intercurrent bronchitis with swelling of the mucous membrane, or the plugging of a tube with mucus. Râles which are bilateral may be ascribed to intercurrent catarrh, although local phthisis may exist—since the phthisical are very prone to this inflammatory process.

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* Exceptionally friction râles may be temporarily increased during the deep breathing which follows the act of coughing.

† See appropriate chapter.

Collateral deductions may be drawn, if the history of the cough and the progress of the case indicate a preponderance, or the sole dominion of bronchial inflammation at the time of observation. Finally, there is one helpful point of general application. If there are evidences of consolidation, and the râles are small, not very moist, subcrepitant, crackling, and localized, the site of tissue modifications, they are usually due to the softening of cheesy deposit, for it is in the varieties of phthisis involving vesicular tissue that this confusion is most liable to occur. The subject is important, since the prognosis and treatment depend largely on the decision of the observer.

In *incipient textural* modifications in the lung—early phthisis—it is well to await the advent of crackling, or some species of moist râle, before deciding that the evidences of percussion and auscultation relegate the tissue modifications to some group of phthisis. In these cases very often these râles can only be excited by the act of coughing. Subcrepitant râles associated with hypostatic congestion, due to the exudation into the finer bronchial tubes of serum, are heard bilaterally, but are differentiated from bronchitis by their association solely with posterior and basic portions of the lungs, and by their association with special states;* from true pneumonia, by their association with these special systemic states, but also by the absence of sufficient dullness or bronchial breathing. Râles of some finer order are associated with atelectasis, which condition is recognized by the distinct association of atelectasis with catarrhal pneumonia, capillary bronchitis, or pressure on air passages leading to involved district. For appropriate evidence by auscultation and percussion.†

Hemorrhagic infarction, during the stages of softening, can naturally give rise to many species of râles. Diagnosis is made by the association of pathological condition with the causation (*see* works on general pathology).

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* If the patient is able to permit respiratory percussion, the greater clearness of sound on percussion during held-inspiration will separate hypostatic congestion, and possibly the other conditions, from true consolidation.

† *See* pages treating of the diagnosis of hypostatic congestion by auscultation *or percussion*.

Cavities.—When these are filled with fluid, naturally all kinds of splashing sounds, from small to large gurgles, can be heard. Similar râles are sometimes audible in bronchorrhœa. The diagnosis of cavities does not hang upon the species of râles heard. If bronchiectatic cavities exist they may be recognized and differentiated by signs already stated. *Finally, simple dropsy* (œdema) of the pulmonary tissues can give rise to râles which will be fine and moist, since they are generated in the finest bronchioles, or even may be developed in the intervesicular tissue during the inflation of the lung. These râles are stamped with their true import by remarking that they are associated with heart disease, valvular or textural kidney diseases, or by the association of dropsy of the general cellular tissues, or effusion into the serous sacs of the body, or with tumors causing pressure on the respiratory tract.*

A moist crepitant râle can be heard at times in states of hypostatic congestion or œdema. The râle is often indistinguishable from a subcrepitant râle developed in the finest bronchioles. There is also one point which will bear reiteration in this place, viz., that pulmonary râles may be heard in inspiration and expiration; in doubtful cases, insist on full inflation (often to be secured only by requesting the patient to cough) before giving an opinion.

In acute consolidations:—

First.—Croupous pneumonia—The dry crepitant râle is heard during the initial states of consolidation, but *invariably* disappears when consolidation is complete.

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* These finer râles are the râles most apt to be confounded with moist pleural frictions.

One must auscult very early in the case to secure this râle, but sometimes the area of pneumonia extends, and then crepitant râles precede the involvement of new districts of tissue. (*See* Pleurisy, for diagnosis of pleuro-pneumonia.) Study especially the general symptomatology, with the physical diagnosis, in this disease. This is very important in the aged, in whom the thermometrical record is apt to be low. In these cases the *advent* of the râles, and the associated signs by auscultation and percussion, must be studied, as well as the immediate results of a physical exploration. The acute catarrhal pneumonia, with its rapidly developed diffused areas of consolidation, is always associated with a widespread development of subcrepitant and mucous râles. It is a disease which not infrequently occurs in children, and is associated with a collapse of neighboring pulmonary districts. (*See* Capillary Bronchitis.) In adults it is also always linked with descending bronchitis, but is more frequent as a subacute or chronic process, as the forerunner of some form of phthisis, or it may occur in the course of emphysema.

The space at our command renders it impossible to enumerate all the conditions favorable to the outbreak of râles. The hints given we intend as suggestions to induce the reader to fix his attention on the natural history of râles rather than on the isolated cause of their genesis.

The râles not associated with some of the groups of phthisis or œdema are the râles in which pathological change is solely confined to the bronchial tubes. The division, of course, relates primarily to *acute* and *chronic* bronchitis. In acute bronchitis the sonorous râles are developed in the larger, the sibilant râles developed in the smaller tubes, and are occasioned by the swelling of the mucous membrane lining the tissues concerned, or the presence of tenacious mucus. After the initial stages of congestion are passed exudation is more fluid, swelling more or less rapidly subsides, and the various grades of mucous râles appear. The sibilant and sonorous râles become scattered and less numerous, and finally give place altogether to the moist râles. In the chronic phases of this disease the sonorous and sibilant râles may be associated with the mucous, since there may be more or less permanent bronchial thickening or transient plugging of the tubes. Râles indicative of bronchitis are said to be always bilateral. This depends on whether the bronchitis is simple or is associated with some form of phthisis or conditions affecting tissues other than the bronchial. The proposition then is: in simple bronchitis, acute or chronic, the râles are bilateral, since râles indicate simply fluid in the bronchial tissue. If other processes are associated, differentiation is based by comparative study of symptomatology and physical examination. In inflammation of the bronchial tissues *only*, the other evidences furnished by auscultation are harshness of otherwise normal respiratory murmur, perhaps even broncho-vesicular breathing, produced by the changes in the bronchial mucous membrane. There is never the concentration of quality nor the elevation of the pitch indicative of condensation of the pulmonary substance. Percussion is negative. The resonance is normal, or presents such slight elevation of tone as to be insignificant.

Vocal fremitus and resonance are unchanged from normal standards described. In conclusion, we again advise a careful study of those cases in which the fine mucous râles are present, especially in cases of bronchitis complicating emphysema.

Ascertain whether they are indicative merely of bronchitis, or whether they indicate patches of catarrhal consolidation. Study chiefly the expiratory sound of the respiratory murmur. This is *low* pitched in simple bronchitis, but high pitched in proportion to the amount of consolidation. The quality of the respiratory murmur becomes concentrated, converting the breathing into the bronchial type. In slight emphysema these changes may be recognized by auscultation long before percussion is available. This is owing to the extensive distribution of the vesiculo-tympanitic resonance. But a trained ear and the use of light percussion will render percussion nearly as useful as auscultation, and respiratory percussion is very useful. (*See* section, Respiratory Percussion.)

Capillary bronchitis is a disease of the two extremes of life, childhood and old age.

The exudation occupying the finer bronchial tubes, we have two very necessary results, excessive dyspnœa, and the finest bronchial subcrepitant râles, and in these two particulars we find the cardinal difference from *ordinary acute bronchitis.*

In children its advent is often so sudden that some laryngeal affection is suggested, such as croup or œdema, but auscultation will speedily prevent this mistake.

It is apt to be confounded with and followed by acute catarrhal pneumonia, especially if it occurs as a complication of measles, pertussis, or chronic catarrhs. We must say that it is often impossible to distinguish it positively from catarrhal pneumonia. We refer to the chapter on physical diagnosis of that disease, only adding that capillary bronchitis is attended by a lower thermometrical range than the catarrhal pneumonias.*

In *adults*, the chief source of error will arise from the confounding of this disease with œdema, or hypostatic congestion.

Our suggestion again is that a diagnosis is materially aided by a study of the temperature, which is higher in bronchitis than is commonly the case in the above processes. Again, a diagnosis may be made by excluding the predisposing causes to œdema or hypostatic pneumonia.

So far as hypostatic congestion is concerned, these have been already stated.

Œdema, if acute, is sometimes the result of sudden congestion of the lungs. It is more commonly chronic, and consists of effusion of fluid into the finer bronchial tubes, and the pulmonary tissue generally. It is associated with dropsies elsewhere as the result of organic disease of heart, kidney, or liver; in frequency, about in the order which the organs have been named. Its prominent characteristics are, in addition to the finer râles, the expectoration of frothy mucus, and greatly embarrassed respiration with intense congestion of the venous system, evidenced by the bluish lip, the feeble or noisy breathing. In acute œdema death speedily terminates the scene. Chronic œdema may be persistent, and so are the symptoms.

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* In capillary bronchitis the bronchial tube leading to a district of tissue may be suddenly plugged. Atelectasis may occur, and in a few hours the case may assume an aspect of greatly increased gravity. In diagnosis consider the sudden development of the serious symptoms; especially dyspnœa. By inspection, note the absence of full thoracic expansion on the affected side, possibly the increased inward movement of the intercostal tissues at the base on inspiration. Inspiratory inward movement of intercostal tissues occurs from obstruction of a bronchial tube by pressure of a tumor, in some cases of emphysema, in pleurisy, and in some cases of vesicular emphysema linked with chronic phthisis. These conditions are elsewhere considered. Auscultation is not so helpful. Respiration may be feeble, absent, or more or less bronchial. Percussion, see Catarrhal Pneumonia.

Asthma.—By asthma is meant a spasmodic narrowing of the bronchial tubes, by contraction of circular muscular fibres. It is idiopathic without structural pulmonary disease, or symptomatic. Both forms are associated with all varieties of sibilant sonorous râles, and after a longer or shorter time by all sorts of mucous râles. These mucous râles are partly the result of congestion of bronchial mucous membrane without indicating inflammation. In this case they disappear soon after the paroxysm is past; or they indicate the association of bronchitis with the spasmodic attack.

The *dyspnœa is expiratory in rhythm.* It is true, there is an obstruction to the entrance of air into the lungs. But in the presence of a case we notice the chest is fully distended. The appropriate expiratory efforts are, however, inefficient, partly from the difficulty in expelling the air, owing to the spasmodic contraction of the bronchial tubes, partly from the lack of stimulation to expiration on account of imperfect inspiration. The evident result is the apparent exclusively expiratory dyspnœa. The lower parts of the chest are fixed and altogether immovable. Few or hardly any abdominal respiratory movements occur, and since the diaphragmatic movements cannot avail, no such movements are made, and the diaphragm remains depressed. It follows that the respiratory murmur is weakened, inaudible, or muffled.

The heart sounds also become very weak, indistinct, and distant.

It is desirable to group the instant features of the attack: First—Suddenness; it often occurs at night; a patient may wake out of sleep with a fit of the disease on him, although a feeling of gradual suffocation may precede the attack. Second—Dyspnœa, intense; the patient desires the windows raised, or walks about gasping for breath. Third—The skin is cold and moist, the visible parts of a pallid or bluish hue, showing imperfect aeration of the blood. Fourth—The attack may last hours, and disappear suddenly, or for days, ameliorating during intervals, to again grow worse, at last terminating gradually. If it is symptomatic, the following are predisposing causes, although the exciting cause is obscure: it is associated with cardiac lesions, such as fatty degeneration, sometimes with valvular disease. It is very commonly interlaced with emphysema* and with chronic bronchitis. The diagnosis of idiopathic or symptomatic asthma consists in its paroxysmal character. Œdema and spasm of glottis differ in not being associated with wheezing. Laryngeal paralysis can be detected by the *mirror*. There is also aphonia, but the stridulous breathing is inspiratory, and there is an absence of adequate chest symptoms.

The pressure from tumors of mediastinum, notably aneurisms, cause symptoms similar to asthma by pressure on the pneumogastric nerves or on the bronchial tree. The diagnosis is only thoroughly established by a diagnosis of the nature of the tumor. To know the difficulty may be half the battle. Asthma acts as a cause of emphysema in early life. Asthma is also a sequel of emphysema.* (*See* chapter on Emphysema.)

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* *See* clinical lecture on Dyspnœa a symptom of intrathoracic pressure, *Med. Times*, Philadelphia, Oct. 11, 1879.

Renal Asthma.—The asthmatic picture due to kidney disease occurs with these two differences, viz., the pulse is very rapid, without cardiac disease to account for it, and the air enters freely the lungs. The asthmatic symptoms are traceable to the difficulty experienced by the blood in reaching the air, due to a spasm of the arterioles of the lungs, instead of an obstruction to the air reaching the blood. The diagnosis consists by exclusion. 1st. Demonstrating the absence of any pleural effusion or other pulmonary cause for the dyspnœa. 2d. Demonstrating the absence of heart disease. 3d. By establishing the existence of renal disease, especially those forms most intimately associated with preponderating interstitial changes. As an important auxiliary to establishing the diagnosis of this form of renal disease by physical diagnosis, note the hypertrophy of the heart, and the accentuation of the second sound.†

A disorder of respiratory rhythm is the so-called "Cheyne-Stokes" respiration. It consists in at first short, then deeper and more and more labored, respirations, until the paroxysm is at its height; then becoming shorter and more and more shallow until the breathing is suspended.

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* True asthmatic seizures may both produce and be produced by a disease of the heart. But what is often called cardiac asthma is not always a spasm of the bronchial tubes. It is usually only a temporary increase of the dyspnœa, dependent upon a decided obstruction to the circulation in the lungs, and not accompanied by wheezing. (*See* Da Costa, Medical Diagnosis, page 257.)

† Dickinson "states that hypertrophy of the heart occurs in seventy-four per cent. of cases of granular nephritis; page 310 of his Pathology and Treatment of Albuminuria." Hypertrophy may not occur, or it can disappear if the vital activity is reduced below the plane of nutrition capable of resulting in *physiological* tissue growth.

The pause lasts from a quarter of a minute to a minute, when the respiration begins again in the same manner, first faint, then a little stronger, then again subsiding in a descending scale, to end in the same stand-still. This kind of breathing is a very bad sign. It is apt to happen when, from some cause, the supply of arterial blood is cut off from the brain or respiratory centre in the medulla. It is rare in diseases of the lungs, much more common in fatty heart, in diseases of the aorta, in tubercular meningitis, in affections implicating the medulla oblongata, and in uræmia.

The above is an excellent summary of our knowledge of this phase of respiration; we would add, that in apoplexy at any site this sort of breathing may occur.*

Sansom summarizes the cases in which this sign is observed as follows:—

"1st. Cases attended with cerebral lesions, viz., cerebral hemorrhage, tumors, uræmia, shock from surgical injury, alcoholism, acute renal disease, tubercular meningitis.

"2d. Cases attended with lesions of the heart or great vessels, viz., fatty degeneration, pericarditis, atheromatous, disease of aorta, aortic aneurism, valvular disease (double aortic, mitral stenosis, dilated aorta coexisting, aortic regurgitation and obstruction). Sclerosis of coronary arteries.

"3d. Cases of certain acute febrile disease, viz., diphtheria (Hütterhenner), typhoid fever, (Wharry). A large portion of cases occur in the male sex, and at a period of life over fifty, when degenerative changes are common, the exceptions being in the acute diseases which I have noted. I consider that the initial lesion is paresis of the respiratory centre, and though this paresis *may be* produced by reflex nerve influence, it is usually a direct exhaustion from cerebral causes."†

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* Da Costa's Medical Diagnosis.

† Sansom's Physical Diagnosis of the Heart, page 36.

Acute Miliary Tuberculosis.[*]—There is much dispute over the proper meaning of the above title. Isolating the true pathology from the confusion, let it represent that general acute eruption of miliary tubercle, which is distributed in and around the *tubular* structures of the body. In the lungs, in the sheaths of the arterioles, in the sheath of the peribronchial filaments, and in the lymphatic vessels. In this disease the distribution of the tubercle is very widespread throughout the body, including liver, spleen, bronchial and mesenteric glands.

The membranes of the brain enjoy an immunity in the *adult*, quite the reverse of the history of similar cases of tuberculosis in the child. The intestines, too, may escape, or even if involved, death happens before much if any ulceration occurs. This is an interesting fact, since it explains why diarrhœa is not a symptom; if, however, phthisis end in general miliary tuberculosis a follicular catarrh or ulceration of the bowels is frequently found quite sufficient to account for a possible diarrhœa. In the lungs themselves the tubercle presents itself in the localities indicated; studding them with the translucent gray granulation alone characteristic of miliary tubercle. At no point do we find the tissue of the lungs consolidated; on the contrary, they bulge with air and yet they do not collapse, showing the tissue is more condensed than natural, and the lung feels as though infiltrated with fine particles of shot. In these cases the tuberculosis has developed itself from some caseous centre in the pleura or elsewhere. In other cases there may be in one or other apex a small, or perhaps a large, cavity. On examination the retraction of the adjacent lung, and the proliferation of connective tissue in the neighborhood of the vomicæ, all show that cicatrization has progressed considerably; these lesions are the result of prior inflammation. There will be, perhaps, more or less evidences of prior pleurisy; often there will be recent pleurisy, the result of recent tubercle. Now the effect of the tubercle deposited in the lungs as described diminishes the calibre of the arterioles, the aeration of the blood is less perfect; a point to be kept in view for subsequent reference.

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[*] *New York Medical Record*, January 22, 1881, page 94.

Physical Signs of Percussion.—All over the chest, on both sides, a vesiculo-tympanitic resonance is audible, sometimes in patches the size of a silver dollar; slight dullness can be developed, anteriorly or posteriorly, but this is not constant.

Auscultation suggests an exaggerated respiratory murmur, with feeble expiration, of the type heard in emphysema, or in supplemental action of a healthy lung when its fellow is disabled; nowhere is the breathing bronchial. With this you can hear, on inspiration alone, numerous fine, slightly moist râles, similar to the crepitant râles of pneumonia, but moist. Moreover, they can be heard at both apex and base of both lungs. They also remind one of the capillary râles of bronchitis, but capillary bronchitis is attended with copious expectoration, and is apt to be associated with the chest and heart alterations of emphysema, or the preëxistence of Bright's disease, etc.* Sometimes dry sonorous and sibilant râles can be heard. If there be cavity at apex it is diagnosticated by appropriate phenomena. Let us not be misunderstood. We acknowledge the existence of tubercular chronic phthisis, also of acute tuberculosis, as complicating the causes of advanced cases of chronic phthisis, catarrhal and tubercular. We now speak of the general acute outbreak, with few, slight, or absent chronic phthisical lesions. The pathology of the disease sheds much light on the physical signs. The development of the râles just described occurs from the irritation of the bronchial mucous membrane and consequent moderate catarrhal exudation into the finer bronchial tubes. If there are friction râles they will be dry, and perhaps some restriction of the movements of the chest will occur by comparing the sides, but both lungs, nevertheless, expand and retract. The dry pleurisy is especially indicative of a tubercular pleurisy. In reflecting on the quality of the percussion resonance and respiratory murmur, remember that the vesicles are hyper-distended, as in emphysema. The respiratory act is imperfect, because of the obstruction to the exit of air, due to the catarrhal secretion in the finer bronchial tubes. The physical signs do not indicate consolidation in any of its forms, and the tout ensemble of the case does not indicate the ordinary forms of bronchitis. Let such physical signs be appropriate to acute miliary tubercle when combined with the general symptomatology. Considering these as briefly as possible, the cough is not attended with much expectoration, without hemorrhage; if there be any sputa it is frothy mucus, small in amount. The scanty sputa is readily understood when we reflect that the amount of bronchial *catarrh* produced by the presence of tubercle is slight.

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* *See* chapter on Emphysema.

The fineness of the râles is thus explained, especially since pneumonia is out of the question as well as œdema.

General Symptomatology.—Sweating is a most marked symptom; it is almost uncontrollable; it is so profuse as to drench the clothing of the patient and the bed in which he lies. The sweating and condition of the skin has always reminded me of the sweating of the death agony, and possibly a similar explanation will serve us. The pulse is a most salient symptom. It is very rapid—perhaps 144 per minute—and this rapid beat is a sustained feature during the entire course of the disease. It is never, in typical cases, below 120, and varies between this and 144 to 150. This persistently rapid pulse is a fundamental symptom of the malady, and is most unvaryingly present. Many writers allude to it; all do not give it the diagnostic prominence here assigned. The pulse is also small; often it is thready. The temperature record is of great value. The range is from 100° F. to 103½° F. during the disease. In the week before death (for this disease is fatal) the temperature may fall to 97° F., to rise in the evening to 101° F.; but daily in that week it will be found below 99° F. This is very constant. Another valuable symptom is in the dusky hue of the face; at times it is quite flushed, as in typhoid pneumonia; at times it is quite pale. The flush is seen often to come and go in change of position. The general surface of the body in the later stages of these cases is also dusky, and often the appearance of the patient will forcibly remind one of the cadaver after death from a blood disease. Perhaps the interference with the pulmonary circulation, due to the distribution of the tubercles, may explain this appearance of the body, and also the frequency of the pulse.*

There is always more or less dyspnœa, apparently without adequate pulmonary lesion other than the tubercular distribution. Possibly, this distribution may explain the symptom. The duration of miliary tuberculosis is from six to nine weeks, usually the shorter period. Especially search for evidence of prior suppurative inflammation. Examine the glands of the neck; note if scrofulosis or consumption are hereditary in the family of a patient, for the connection between these conditions and the disease is very intimate. Some forms of prior inflammation, usually in a cheesy state, forms the nucleus for absorption and the lighting up of tuberculosis at the smallest provocation.

In cases of general tuberculosis complicating consumption, the physical signs of the antecedent phthisis often mask the none too characteristic physical signs of the presence of the complication. A similar symptomatology, however, prevails, but with modifications induced by the presence and preponderance of the catarrhal inflammation. First, the pulse may not be so persistently frequent, unless suppuration has produced exhaustion; even then it is not significant of tubercle, because it is customarily observed in cases of hectic. The temperature range is often higher, and corresponds with those given by Wunderlich and Niemeyer. These temperatures, it seems to us, are associated with acute miliary tubercle, complicating advanced inflammatory phthisis. In these cases life is prolonged until cheesy metamorphosis of the tubercle has occurred in addition to the antecedent older cheesy material. The elevation of temperature is probably due to the suppuration. On the contrary, in acute miliary tuberculosis death happens before much or any cheesy degeneration occurs. The sweating, however, is present—not hectic sweats, but long continued, almost uninterrupted, uncontrollable sweating. By treatment this may be modified, but the symptom is of primal importance.

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* The frequency of the pulse in renal asthma is owing to a similar cause.

In secondary tuberculosis the dyspnœa is not so great, since the more chronic antecedent malady has reduced the systemic demands for oxygenated blood; and although the acute development of tubercle may cause some shortness of breath, it is not so definitely manifested as in the primary disease. In acute primal miliary tuberculosis there is no emaciation or evidence of the wasting of consumption, always present in the secondary form. Finally, we repeat: metallic râles, some species of cavernous breathing, vocal resonance, or tympanitic percussion, will indicate a cavity.

The Differential Diagnosis consists in a differentiation from typhoid, remittent, or typhus fevers. In the first, the contrast is seen in rapid pulse early in the case, without the typhoid state, the cyanosis, the physical signs in the lungs, the sweating, the absence of characteristic rise in temperature, and the absence of prodromes from the history. From remittent, by history and therapeutic tests. From typhus. The ensemble is somewhat like many cases of typhus, for in this disease there is the early rapid pulse, a dusky hue of the skin. The history of the case, a minute analysis of the pulmonary symptoms, the typhus eye, the environment from which the cases are developed, are perhaps the safest indications for diagnosis by exclusion, while the eruption, if it be characteristically present, is conclusive. It is only, of course, in large seaboard cities where this disease can be a source of confusion.

The other form of tubercular disease of the lungs is that which originates a larger or smaller class of cases of primary phthisis in its earlier or later stage; in order to avoid controversial ground, we quit it with the statement: We have been led to devote the space occupied by this discussion of tuberculosis, since we have had unusual opportunities for the clinical observation required for the above sketch; and the outline of the subject, from its importance and frequency, cannot be too sharply defined.

CHAPTER X.

DIAGNOSIS OF THE LESIONS AFFECTING THE PLEURAL CAVITIES.

In a logical approach to a diagnosis of lesions under the above heading, it is fundamental, first, to consider in series the pathological conditions affecting the pleural cavities, and, second, the effect of these conditions upon the lungs as air-containing tissues, and on the methods resorted to as means of diagnosis. We are confronted at the outset by the various forms of pleurisy, with and without effusion. The effusion may completely fill the pleural cavity. Between this condition on the one hand, and on the other those cases of pleurisy in which the effusion is very small—scarcely more than covering the floor of the cavities—we have every possible gradation in amount of effusion. We have also two species of inflammation, attended by the formation of liquid or of solid material; the terms serous and fibrinous have been applied to describe them. If the formation be simply serous, the fluid will gradually accumulate, like water in a well, until the entire chest is filled. If the effusion be fibrinous, it may spread itself very widely over the surfaces of the pleura, until they are more or less completely coated with flakes or layers of lymph. We can probably approach this subject most readily if we state as illustration that we can have, in the first place, an acute pleurisy which is capable of terminating—

1st. In cure, or
2d. In chronic pleurisy.
3d. Pleurisy with effusion, acute or subacute.
4th. Empyema.

It will be in order very briefly, and perhaps somewhat diagramatically to state the pathology of these lesions, to furnish a basis for discussion of their diagnosis. We shall also then have a series of standards to which conditions producing similar effects can be referred and compared. In acute pleurisy we are aware that the effusion may be serous or fibrinous. If fibrinous, plastic lymph more or less moistened with serum, is distributed over the pleural surface in flakes, which are more or less thickly massed at some points, while at others the pleura may be normal; there is also always more or less fluid effusion, in which flakes of fibrine float. In the initial stages there is more or less congestion of the pleural vessels, both of the visceral, and parietal pleura. It is important to recall the intimate association of the blood supply of the visceral pleura, with the blood vessels in the polygonal spaces, seen on the visceral layer of the pleura, since they represent the bases of the pulmonary lobules at the surface of the lung. The result of this is to often form vascular projections or vegetations, over which, if the process proceeds to exudation, the lymph is most thickly coated. But the vascular congestion possesses this further interest, viz., it exhibits the intimate relation of pleural to pulmonary congestions and inflammations. The amount of serous effusion may be large compared with the amount of fibrinous, or the reverse may occur. The aggregated *fluid varies from* half a pint to a pint and a-half; this amount requires several days to accumulate, perhaps a week or more. Occasionally the effusion continues to increase until the pleural cavity is quite filled. We have classified this as acute or subacute pleurisy with effusion. The distribution of the pleurisy is almost always unilateral. A bilateral pleurisy indicates a systemic etiology, such as measles, scarlatina, typhoid and typhus fevers. It may also be caused by Bright's diseases, gout, rheumatism.

Finally, it may follow wounds of the thoracic wall, or one lung may be already the seat of phthisis. Bilateral effusion belongs, also, to some of the classes of dropsy (hydrothorax). It is next in place to consider the effects of these changes upon the lung beneath. These vary in acute disease in accordance with the amount of effusion present. If this is very moderate, and the pleurisy chiefly fibrinous, the lung will be moderately compressed, its complete inflation will be resisted, and there will be a more or less thickened vascular pleura interposed between the visceral and costal surfaces, so that the air-containing lung is further removed from the ear than is physiological. If there be a pint or more of liquid within the chest, the lung will be borne upward, and there will be a tendency to compress the pulmonary substance backward. The vesicular tissue is chiefly encroached upon; the bronchial tissues, being mere resistant, remain nearly as thoroughly patulous, and capable of transmitting air as normally. Let us fancy, however, the pleurisy, instead of terminating in resolution and absorption at the above stage, passes on into the stage of large effusion. The effect upon the lung must be considered under a twofold postulate:. 1st.

The effusion, if very large, may positively compress all the pulmonary tissues, including the bronchial, until the air is absolutely expelled, and the lung itself is flattened into a small space against the vertebral column, occupying mostly the scapular region, or a space sometimes not larger than the palm of an ordinary-sized hand. 2d. The fluid may force the lung more forward and upward than backward—so that we may outline it anteriorly, perhaps, so low as the second rib; posteriorly, the area occupied is not so large. In either case the lung tissue must be considered as sustaining very abnormal phases of tension and elasticity. If the bronchial be the only tissue permeable to air, then the pulmonary atmospheric tension must be high, and the elasticity nearly abolished. If some of the vesicular tissue remain pervious to air, still the tension under which the air is retained will be increased, and the elasticity of the tissues will be lessened, inversely, according to the increase of the fluid. Finally, a pleurisy with effusion will alter the relations sustained by the thoracic viscera to the chest surface; if we are aware of the size of the areas occupied by these organs, we can readily detect them in whatsoever situation they assume.

Acute Pleurisy Diagnosis.—Inspection is not always possible in private practice, nor is it so essential to a diagnosis, as is auscultation. But the effect of the above changes, as manifested to the eye, are a notable lessening of respiratory movement, due not to the lesions so much, as to the pain caused by the respiratory movements, which are not, therefore, attempted. But there is also a decrease in the movements of expansion and retraction of the affected side. It is not true, as often stated, that respiratory movement is abolished; on the contrary, there is under almost all circumstances an up and down movement of the ribs on the affected side, which betokens the elevation of the ribs in common with the non-affected side; but expansion and retraction are subtracted.*

Increasing Effusion.—The more fluid contained within the chest, the more distended will the affected side become; the interspaces will become filled out to a level with the ribs—an increasing want of expansion and retraction—although, even when the chest is fullest, there is more or less up and down movement. By inspection, if the effusion is on the left side, we note that the heart is displaced to the right, proportionately to the amount of effusion; the apex may come to pulsate in the epigastrium, or entirely to the right of the sternal bone—even in the right axilla, anywhere from the second to the sixth interspace, the whole ventricular outline may be bounded by percussion. The pulsation to the right is often due to the shock of the entire ventricle against the ribs. On the other hand, similar displacements of the heart to the left side, occur in right-sided effusions, only the displacement is not so great.†

Mensuration.—The remarks made in the section on emphysema with reference to unilateral enlargements of the chest prevail, with this additional remark: The tracing of the contour of the chest on paper will show a departure from the physiological shape, although the

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**See* section on Modifications of Respiratory Rhythm, and contrast intercostal neuralgia with pleurisy.

† Displacements of apex-beat are a notable symptom of chronic pleurisy of fibroid phthisis, and in the various diseases of the cardiac valves—conditions readily differentiated from pleural effusion. (*See* also section on Emphysema, for summary of epigastric pulsations.)

circumference in inches may or may not be larger upon the side of effusion. The fact remains, however, that in many cases the side of the effusion will exceed the unaffected side by measure.

The unaffected side will be enlarged to a greater or less extent, since it is practically in a state of supplemental emphysema, but the interspaces will not be bulged. It is wonderful to note the degree to which toleration of large effusions may occur, in some cases without causing noticeable dyspnœa. The rapidity with which the fluid accumulates in pleurisy, with effusion, bears a direct relation to the amount of dyspnœa. So we may set down for a clinical fact, that complete filling of one pleural sac may occur gradually, *without* noticeable dyspnœa. *On the other hand*, the respiratory efforts on the unaffected side may be painfully exaggerated. In the initial stages of a pleurisy with effusion, there is often much dyspnœa from the pain caused by efforts to secure complete respiratory action. The distress seems greater than in many cases of pneumonia.*

Exceptional Physical Signs.—In some cases in which an effusion nearly fills the side of the chest, but especially in cases of lesser effusions, and those of subacute or chronic type—particularly when they are undergoing absorption—the following may be noticed: an actual in-and-out movement of the tissues of the interspaces may occur in the involved side, synchronous with respiration. Sometimes with *inspiration*, a depression of the intercostal tissues occurs. (*See* Edinburg *Med. Journal*, Oct., 1880.) There is frequently no obliteration of the intercostal spaces, and, as we have said before, no increase by measure of the affected side. But further than this, the side of the chest supplementally active in respiration may be prominent.

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* See Intercostal Neuralgia.

In the paper in the *Times*, referred to below,* a full discussion of the causes leading to these phenomena will be found; the conclusions there arrived at are as follows: The longer a fluid effusion persists, the more dense does it tend to become, and the area it occupies is diminished, just as a jelly when it forms occupies a slightly smaller area than the fluid out of which it was formed, and thus the intercostal spaces tend to sink inward in this class of cases. In addition, consider that during the time an effusion persists the chest walls are bulged beyond a physiological position; and from this point, owing to inherent resilience, they tend outward at each inspiration, but the lung elasticity is abrogated, to a varying degree, by the compression of the pulmonary tissues by the effusion. Thus it comes to pass that the state of the chest walls resembles their condition in moderate emphysema; consequently, the pulmonary intercostal tissues are depressed on inspiration, and tend to puff out or return to their morbid physiological state during expiration, just as these tissues act in emphysema of the lungs.

Movements of the Intercostal Tissues.—There are two other explanations of movements of the intercostal tissues during respiration worthy of notice, viz., in the first place, the union of the pulmonary and costal pleura by adhesions; but a notable point of difference between this and the above is, that the retraction of the tissues would occur in this case during expiration, not inspiration. The tissues may retract on inspiration, in cases of pressure or obstruction on some portion of the bronchial tree, but this cause of pressure need only be kept in mind, since it is easily determined.*

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* Philadelphia *Medical Times*, May 21st, 1881.

The greater enlargement of the non-affected side is to be explained by the fact that the vicariously dilated lung tissue has not sufficient elasticity to oppose to the eccentric resiliency of the chest walls.

On the affected side, then, the accumulation of fluid gives a more rotund appearance to the chest walls, but by measurement the circumference is frequently less than the healthy side, although the effusion is present; and this difference in size is abetted by the tendency of the chest walls to retract, if inadequately supported from within.

Palpation.—This method affords a most unqualified means of diagnosis of pleurisies. The waves of sound are prevented from radiating to the surface of the chest wall, by the separation of the pulmonary tissue from the chest wall, implied by this disease. This is quite the reverse of the findings in cases of consolidations of the lungs, in which the vocal fremitus is increased.

The evidence of palpation is always valuable, whenever the tone of the voice is sufficiently sonorous to develop any fremitus on the unaffected side. With these premises, *the absence of vocal fremitus is a reliable sign of pleurisy—the more pronounced the effusion, the more positive is the sign.*

Percussion.—In the very early stages of pleurisy, the evidence by percussion may be negative, since the conditions of the pleura, already described may not sufficiently separate the lung from the chest wall to render the sounds positive; the diminished expansion of the lungs, and the increased tension under which the contained air is maintained, all tend to result in dullness; but so soon as effusion occurs, the note becomes flat—differing in this respect from the condensations of pulmonary tissue, in which the sound becomes only very dull—because we develop vibration in the air within the bronchial tubes. The flatness is directly the sequence of the separation of the lungs from the chest wall, by a dense non-resonant collection. Naturally, as the effusion ascends the chest, the percussion resonance tends to become flat in proportionate areas. The sense of *resistance* imparted to the finger used as a pleximeter is very *marked*. Especially are the flatness and sense of resistance pronounced at the lower portion of the chest, since the fluid naturally settles there. The line of flatness is commonly said to be capable of changing its level in partial effusions, with the changes of the posture of a patient, and this is so in all cases in which the fluid is not circumscribed by adhesions. These, however, may limit the effusion into an indefinite number of pouches, of smaller or larger capacity.

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* *See* Capillary Bronchitis.

In these cases, the level of fluid cannot change, and the lines of flatness are unchangeably located. In any event, however, the line of flatness is higher behind than in front.*

Above the Layer of Liquid.—The significance of percussion is best understood by a thorough appreciation of the conditions under which the air yet remaining in the lung is placed, and the condition of the pulmonary tissue. The resilience of the pulmonary substance is more or less diminished, consequently the air is restrained in a state of tension, *varying* with the *size* of the effusion. The resonance, upon principles already discussed, becomes high-pitched in the immediate vicinity of the liquid; the quality may be dull, or some tone of tympany, owing to the compression of the lobes of the lung. Notwithstanding this, the transition from the clear pulmonary tone to the flat percussion, over the area of effusion, is markedly abrupt. The only source of confusion which sometimes arises occurs when some process of phthisis or pneumonia complicates the effusion. The differential diagnosis will be subsequently described.*

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* *See* section on Respiratory Percussion used as a method to define the upper margin of a pleural effusion.

This tympanitic quality is naturally best appreciated in front of the chest, near the superior portion. It may be so high-pitched as to become almost amphoric, and in children, or those with thin and flexible chest walls, the cracked-pot resonance can be developed. (*See* cracked-pot resonance in phthisis.)

One caution must be observed in percussion of small effusions on the right side, viz., not to mistake the line of hepatic dullness for the line of an effusion; in this case, change of posture often resolves the difficulty.

On the non-affected side, the pulmonary resonance is vesiculo-tympanitic.

Auscultation.—The extended reference to the physical signs of pleurisy is made because these signs are the fundamental basis of the diagnosis, the general symptomatology being very misleading. We naturally study the respiratory murmur and the vocal resonance, as affected by the preceding conditions. In the primary stages of pleurisy, the principle changes in the respiratory murmur pertain to rhythm; inspiration and expiration are shortened; the respiratory murmur is feeble, chiefly because of the pain caused by the respiratory act. Associated with the murmur are râles, of the dry or moist variety of friction. Enough has been said, in the chapter devoted to these adventitious signs, to guide, in their separation from intra-pulmonary râles, the concurrent evidence of physical diagnosis, and symptomatology of pleurisy must be invoked to distinguish between sounds often acoustically identical with intra-pulmonary râles.*

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* *See* Respiratory Percussion on this point.

By-and-by, as the effusion ascends in its level, we notice two remarkable results of auscultation. *In the first place,* if the chest be half or completely filled with fluid, the respiratory murmur may be completely abrogated. Paradoxic as the expression seems, it vividly describes our sensations, if we affirm that we hear silence all over the region of the effusion. The results of the auscultation of the voice is in accord with the above. The vocal resonance is cut off and abolished over the region occupied by the fluid. This is manifestly because no air can enter the chest at the situation of the effusion. *Above the level of fluid,* the prevalent conditions described, when speaking of percussion, have prepared us to recognize the murmur as exaggerated or broncho-vesicular, according to the degree of compression of the vesicular substance. In many cases the respiration at the apex, on the affected side, anteriorly, may simulate the amphoric. To guard against mistakes

* Deviations in respiratory rhythm for diagnosis of pleurodynia, p. 185.

on auscultation or percussion, consider the abrupt line below which respiration ceases, or percussion resonance becomes flat, a line so abrupt that the transition is often best appreciated by the stethoscope. Consider also the associated symptomatology, which is very distinctive in phthisis; besides, the cavernous physical signs are less defined.

In the second place, we may have a widely different result of auscultation. The respiratory murmur may be bronchial or tubular, almost identical with the breathing in pneumonia.*

How shall we differentiate? Let us note that, in consolidation of the lungs, the tubular or bronchial breathing has a brazen ring, which carries with it the impression of superficialness of origin. On the other hand, the plane at which the bronchial breathing of pleural effusion is developed, is manifestly deeper. The sound is more distant and muffled, giving the impression that the respiratory murmur is being carried on at a distance. This impression is more readily conveyed by illustration at a bedside, than can be stated in language, and it is more easily grasped in the clinical study, than might be fancied from the description. The vocal resonance shares in the modification. It is not cut off, but may be heard very loudly as bronchophony. At the same time, it is separated from the bronchophony, indicative of consolidation, by the distinction mentioned as safeguards in the recognition of the respiratory murmur. Near the spinal column, posteriorly, the bronchial breathing and the bronchophony are very constant. This is, probably, because, in a majority of instances, this is the situation of the lung. In explanation of these physical signs, the following is apropos:—

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* The most common form of consolidation is pneumonia, but sometimes phthisis.

The class of cases in which the above physical signs are observed, comprise, as a rule, those in which the effusion partially fills the chest; the air which enters the lung above the fluid generates, for obvious reasons, bronchial breathing, and the murmur is transmitted downward—over the area of effusion. In cases of sacculated effusion, the loculi being small, the conditions of condensation and tension, brought about by the bands of lymph, may favor conduction; in these cases, the respiratory murmur is heard as a bronchial sound. Carefully going over the chest with a stethoscope, will oftentimes enable the observer to note areas over which the murmur is much more distant, muffled or absent, than others; these spots so selected are often the areas of effusion.

On the whole, whatever be the real reason for the persistence of bronchial breathing, the most useful distinguishing feature is the element of distance, or muffled quality of the murmur. Now the persistence of the *vocal resonance* is explicable upon the same grounds; but its increase above the line of a pleural effusion is attributed by Dr. James (*see Edinburg Journal*, Oct., 1880) not to condensation of the lung above the fluid, but to the waves of sound being transmitted through the bronchial tubes to the effusion and reflected upward, and the ear appreciating the resonance physiologically diffused over the entire thoracic cavity. This increased resonance can be heard at any point of the chest above the effusion. Skoda, however, located it at the lower angle of the scapulæ, near the large bronchi, near the vertebræ, and above or below this line. Guttman declares the clear bronchial vocal resonance is only heard when the dense lung tissue touches the chest, usually between the vertebral column and the scapulæ. The attention to this study betokens its importance; it is the most important supplement to the results of auscultation of the respiratory murmur.

We may likewise notice distinct bronchophony over the region of effusion; the element of distance, or the muffled quality of the sound, is the only element to enable us to make correct inferences.

Physical Signs during Absorption.—We need not pause to indicate the retrograde physical signs of diminishing effusion, or the cure of acute or subacute pleurisy. For a long time after vocal resonance and fremitus have returned, and the respiration is again heard, it continues enfeebled, and its character is indeterminate; it is neither vesicular nor purely bronchial. The friction sounds may reappear as the roughened surfaces again come in contact; finally, the percussion very gradually becomes normal.

Most generally, more or less false membranes unite the two pleuræ, and the intercostal spaces resume their normal shape, and the chest is either restored to its natural size, or is left permanently somewhat contracted. The bronchial breathing near the vertebral column persists for a long time, since a lung that has been compressed unfolds but slowly. The usual general symptomatology, and physical signs of pleurisy, as contrasted with pneumonia, are appreciated by the following table, in the main abstracted from Dr. Da Costa's diagnosis:—

PLEURISY.	PNEUMONIA.
Sharp pain ; not necessarily referred to the affected side.	Dull pain.
Friction sound ; dry cough ; impaired chest motion.	Crepitant râle ; cough followed by expectoration.
In stage of effusion, obliteration of intercostal spaces ; enlargement of side ; displacement of several viscera. Exceptions frequent (see text) ; absence of expansion by persistence of up and down motion of ribs.	In stage of hepatization none of these signs are manifest.
In a large majority of cases, flatness, with absent, or distant and muffled bronchial breathing, and voice signs correspond.	Dullness with marked bronchial respiration, brazen, and near the ear, distinct thoracic voice, that is bronchophony.
Vocal fremitus abolished.	Vocal fremitus increased.
Decubitus is often in the affected side.	Decubitus not peculiar, sometimes on the sound side.
Sputa frothy, rarely any râles in the chest.	Sputa rusty colored, râles from accompanying bronchial inflammation common.
Febrile symptoms usually slight.	Febrile symptoms severe.
Temperature record irregular, not characteristic ; rarely high.	Temperature record much more characteristic. Temperature rises rapidly, soon after onset ; then is continuous, with marked evening exacerbations, from two to three degrees, and morning remission. Often reaches 105°. May show sudden elevations and striking falls in the whole course of the fever. Toward the end of disease generally rapid. High temperatures especially common in pneumonia of upper lobe.

In pleurisy, as in pneumonia, *tympanitic distention of abdomen* may secure an elevation of the diaphragm, and seriously modify the results of *percussion and auscultation.* It is a condition easily recognized, if once suspected; on the left side a drink of water will often settle the question, since the ear if applied, will hear the râle in the stomach.*

Differential Diagnosis.—The above sketch will be incomplete, unless reference is made to the possibility of confounding the severe pain of pleurodynia (that is to say, neuralgia of intercostal nerves or muscles), with pleurisy.

The chief point of similarity is the restriction of the respiratory movements. There are, however, no râles in pleurodynia; there is also only pain, with *tenderness* on pressure, much aggravated by movements of the arm. The pain is of two kinds, either definitely located in the course of the affected nerves, or else, if the pain, if muscular, is fugitive and shifting, attacking often both sides of the chest. The cardinal differences are the association of *tenderness* with pain in pleurodynia. In pleurisy there is pain, but no tenderness. In pleurodynia there is no fever, which is a symptom of pleurisy. *Pleurodynia* is a source of great suffering to *phthisical patients,* and in their case it is of considerable importance to discriminate from a therapeutic standpoint. *Pleuro-pneumonia* is often very difficult to establish, positively, the diagnosis. The physical signs of the two diseases, pleurisy and pneumonia, undergo modifications by the blending of lesions.

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* *See* Introduction, on influence of abdominal states on percussion and auscultation.

The significant physical signs of pleurisy are the muffled respiratory murmur, and becoming more distant in proportion to the increase of fluid. There may be friction sounds in the early stages. On the whole, we are inclined to place great confidence in the absolute flatness of the percussion note over the level of fluid in these cases, since of all cases of pleural effusion, those connected with pneumonia are most apt to present us with a very distinct bronchial breathing, often hardly to be differentiated from that indicating consolidation. Next to percussion, the vocal fremitus is most reliable in its results.*

Pleural Effusions—Hydrothorax.—This term is commonly applied to those effusions in which the accumulation occurs from causes other than inflammatory. In a word, the condition is one of dropsy, and is associated with diseases of the heart, kidneys, liver, or systemic states influencing the crasis of the blood. Important distinguishing points are that it is often associated with general dropsy. Hydrothorax is bilateral; pleurisy, even if bilateral (which is rare), can, perhaps, be antecedently located in one or the other side, or it can be traced to an altered blood crasis, the result of deficient assimilation or elimination. There is also an absence of frictions, or a history justifying the hypothesis of antecedent inflammation. The analysis of other conditions, often mistaken for pleurisy, will be found elsewhere; *e. g.*, pericardial accumulation, enlargement of the heart or liver, intra-thoracic tumor, emphysema or pneumothorax.

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* *See* also Respiratory Percussion.

CHAPTER XI.

CHRONIC PLEURISY.

This division fairly includes the results of prior inflammations. It might also include the states of effusion already described. We shall restrict its meaning to the description of the thickenings of pleural membranes, the result of pleurisy, and to the conditions under which the pleural cavity is divided into compartments by false membranes. A considerable portion of the effusion may remain encysted in pouches formed by these membranes. We also meet with pleurisies chronic from the first, following certain diseases of the lungs. The process may indicate merely fibrous thickening of the pleura in filamentous or membranous adhesions, or the pleura itself may be enormously thickened, and, as subsequent contraction is inevitable, the pleural cavity itself may be obliterated. In chronic pleurisy especially, if there has been pus in the effusion, we may find a more or less complete cuirass enclosing the pleural cavity. The deposit is formed upon both the visceral and the parietal pleura, and is composed of calcareous plates of varying thickness. Some of these conditions of pleural thickening are commonly associated with more or less chronic interstitial pneumonia, and the diagnosis is blended. Pleural adhesions are associated with most of the forms of phthisis.

In the course of chronic suppurative pleurisy, lesions of the ribs, periostitis, necrosis, exostosis, etc., are quite common. To avoid repetition let us say in the outset that the diagnosis of associated interstitial pneumonia is based chiefly on the association of the clinical symptomatology and etiology, with the evidence of physical diagnosis as already described.

Further, that the diagnosis of encysted effusion embraces the features already dwelt upon. Chiefly important are the local prominences, combined with physical signs of effusion, and the history of the case evidencing pleurisy. Remember that these are the cases in which bronchial breathing may be very distinct, the vocal resonance very clear. Search diligently, inch by inch, over the surface of the chest, for the areas over which these physical signs are most distant, most muffled. Make use of the stethoscope, since the ear can take cognizance of the sounds developed over too wide an area. Percuss carefully and it may be possible to develop pulmonary resonance around sites of flatness. The pulmonary resonance is, in these cases, developed from portions of lung tissue, which may be drawn into corners of the chest by the adhesions, and completely surround a pouch of fluid. Bear in mind that these projections of lung, may themselves be involved in a process of chronic inflammation, mostly of fibroid type. On the left side, it is possible to mistake enlargements of the heart for pleural effusion.

The pulsation of an enlarged ventricle is peristaltic, and can be located as belonging to the heart by auscultation of the rhythm of the shock. If transmitted pulsation exists, the pulsation is not peristaltic, nor is it as expansile as in direct beat.* Aneurisms and tumors may cause error; their discussion will be taken up later. Analyses of such cases are among the intricate problems of diagnosis, and require an application of our conceptions of the principles underlying general as well as physical diagnosis.

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* Yet this rule has exceptions (*see Philadelphia Medical Times*, May 21st, 1881, p. 537). Dr. Henry made a few remarks upon a case of pulsating emphysema in which expansile pulsation was an important feature.

The various degrees of thickening of the pleura produce important changes in the perimeter and contour of the affected side. The expansion of the lung, and its elasticity, are more or less modified; the eccentric resiliency of the chest-walls is overcome by the contractile power of the newly-formed tissues. A solid union may be formed between the pleural walls, the ribs may be drawn together and approach one another, especially at the lower part of the chest, so that the diseased side becomes much smaller than the healthy side. The *unphysiological contour* is best displayed by *tracings*, the inferior part of the chest being most altered; such are the results of inspection and mensuration.*

Percussion.—The pitch and quality of the note must vary, in accordance partly with the amount of air permitted to enter the chest, in accordance with the thickness or thinness of the external thoracic walls, and in accordance with the state of the abdominal viscera. It is in these cases most particularly that the tympanitic distention of the stomach or abdominal viscera may mislead.

Auscultation.—Precisely similar conditions influence the respiratory murmur. The rhythm is interrupted, the intensity is very feeble, or, if in the compressed and possibly fibroid lung the bronchial tubes are pervious to air, the respiratory murmur may be bronchial or even tubular, perhaps more or less muffled or distant in quality.* The results of *inspection* are very valuable, since retractions at the base of the chest mean, usually, chronic pleurisy; on the contrary, retractions at the apex denote probable intra-pulmonary processes.

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* Chronic fibroid phthisis singly or combined with pleurisy, may produce similar changes.

Vocal fremitus is absent, but sometimes, if the separation of chest walls from the lung is not very great, and there is a fair pulmonary expansion, some vocal fremitus can be felt. *Vocal resonance* is of variable assistance, and must be considered as corresponding to the respiratory murmur. If the voice is sonorous, the vocal resonance is well pronounced. It is more concentrated and louder than over normal areas, because the adhesions are favorable to the transition of voice, but the voice, however, is more muffled and distant than it would be in a case of intra-pulmonary lesion. *At the base of the lung* it is often very hard to distinguish between slight chronic pleurisy, with or without effusion, and certain kinds of phthisis.

Inspection, as showing moderate retraction and impaired expansion; *percussion*, showing an approach to flatness, rather than the dullness, which might be expected if the phthisis was uncomplicated, are signs which best interpret the respiratory murmur. Usually, the respiratory murmur is of feeble intensity and interrupted rhythm.

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* See Introduction, on abdominal states as influencing physical diagnosis.

CHAPTER XII.

EMPYEMA—PNEUMOTHORAX.

Finally, as stated, pleurisy may terminate in empyema. It may be purulent from the outset, when the pleurisy is the result of infection from purulent foci situated elsewhere. It may originate in connection with phthisis or suppurative pneumonia. It may originate in the parietal pleura, and succeed peri-hepatic inflammations or abscesses of the liver opening through the diaphragm. Wounds or fractures of the ribs also often give rise to empyema. Usually the quality of the pus is laudable, but in infectious pleurisy it has a fetid odor, due to decomposition without perforation into the lungs. Purulent pleurisy may last months or years. The terminations include either evacuation of the pus through an intercostal space; or by perforation of the parietal pleura it may form a pathway to the bronchi; or through the diaphragm into the peritoneum; or pass into the mediastinum, or along the vertebral column as far as the psoas muscle; or into the tissues forming the abdominal wall, and so outward. The termination of spontaneous external evacuation, through an intercostal space, may or may not be preceded by periostitis, with subsequent caries or necrosis of one or more of the ribs, and it is preceded by an œdema of the intercostal tissues. The pleura may be converted into a complete so-called pyogenic membrane. The lung may occupy the positions already described in pleurisy with effusion, or it may be bound down flatly against the diaphragm, or in any position. The substance of the lung may be so absolutely compressed as to be non-air containing.

Physical diagnosis is not very helpful in the recognition of empyema. Contractions or adhesions of the pleura may prevent as much displacement of the heart and diaphragm as in ordinary pleurisy.

Inspection will show a very constantly increased circumference of the chest, and bulging of the intercostal spaces of the affected side.

There is often a unilateral œdema of the corresponding side of the trunk. Dr. James, Edinburg *Journal*, October, 1880, says: "In empyema these signs are due to the more acute pleural inflammation, producing at the same time a greater amount of paralysis, or possibly inflammatory alterations in the underlying intercostal tissues. Thus occurs greater intra-pleural tension through diminished absorption, resulting from an interference with the respiratory movements. The lymph circulation, in the intercostal spaces, is dependent on the movements of the intercostal muscles, just as the circulation in the diaphragm is dependent on its movements. In this way the lymphatic circulation is doubly disabled." Yet this is not invariable, since the condition is present in cases of hemorrhagic, and sometimes in simple pleural effusion. On the whole, however, if unilateral œdema of the chest walls is present, we may not expect a simple serous effusion. The *vocal resonance* is apt to be very distinct all over the chest surface in these cases, possibly because of the presence of the adhesions and thickened pleural tissues favoring conduction, in a great many cases.* When this occurs, careful auscultation will show that at some points on the chest wall the vocal resonance is more muffled than elsewhere. These cases are often cases of sacculated effusion, and these points of muffled vocal resonance offer most favorable spots for paracentesis.

Local surface thermometry may become an available means of establishing the diagnosis of empyema. The writer's experience with this method has been limited to a few cases observed in the last two years. But in these cases the temperature was increased, in five out of six instances, as compared with cases of serous effusion. The case in which no change was noted was a patient in whom the entire pleural surface was covered with a thick deposit of calcareous degeneration, forming a cast of the cavity, and the pleura had been thickened for a long time.

Clinical diagnosis sometimes consists in grasping one or more of the most salient features of a case, and following up the isolated fact as a clue, perhaps capable of disclosing the intimate nature of a complicated condition. In empyema we have a large internal abscess. All internal collections of pus give direction to a group of manifestations, embracing thermometrical variations, sweating, chills, a natural history termed hectic. Clinical observation will perhaps some day differentiate the diurnal wave of thermometry in cases of internal abscess. This has been done in abscess of the liver, in which case the remittent type, and a comparatively low range of temperature, 100° to 102½° F., is the rule. But in empyema the temperature is more variable, rising higher or lower in the thermometrical scale.

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* *Medical Times and Gazette*, January 3d, 1880.

It is the duty of the physician, with the above ascertained facts on the one hand and the physical signs of pleural effusion on the other, to solve the problem, by the exploratory puncture with aspirator or hypodermic needle. At the same time, strange as it may seem, large collections of pus may persist in the chest without much rise in temperature. The cases in which this observation has been made would seem to be those cases of chronic pulmonary and pleural disease in which the pleura are thickened and indurated. Perhaps, in this condition the pleural thickening opposes a sufficient barrier to the development of renewed inflammation from fresh pulmonary catarrhs. The symptoms, as has been said, are so latent that the pus may remain hermetically encapsuled for months or years.

Hæmothorax proceeds, usually, from the rupture of some of the newly-formed blood vessels, in an attack of pleurisy, unless associated with new formations in the pleura. We have no diagnostic symptoms; the operation of paracentesis often first reveals it.

Pneumothorax is a condition consequent, as a rule, upon some prior disease of a lung. This disease consists, sometimes, merely in the rupture of an isolated patch of superficial consolidation which has formed, a small superficial cavity (the rest of the lung being healthy), and by ulceration of its walls air escapes into the pleural sac. More commonly, perforation of the visceral pleura happens in advanced phthisis. "Da Costa gives a rare mode of origin by the rupture of a distended air vesicle." It is also true that air may enter the pleural sac by the perforation of the pleura from without, in cases of wounds. An instance of pneumothorax from wounds, is the passage of air into the cavity of the chest during the operation of paracentesis. The entrance of air is commonly followed by the effusion of liquid—in a word, pleurisy is engendered in some way, perhaps by materials of septic nature contained in the air; however this may be, as the result we have *hydro-pneumothorax*, which embraces the majority of cases; simple pneumothorax being comparatively rare.

The next important step is to ascertain whether the opening from the lung is patulous or closed. Very soon after the fistula is opened from the lung into the pleural cavity, the opening may be closed by inflammation, or the orifice may remain patulous. The disposition of the lung undergoes the same modifications as in pleurisy, save only that pleural adhesions may have formed which will retain the lung in special sites. If there are many adhesions prior to the formation of the pneumothorax, the collections of air may be loculated or pouch-like. The transit of air from the lung into the pleura is attended usually with great pain, together with the sudden development of the most intense dyspnœa. The condition occurs, as a rule, during a paroxysm of coughing, and death may very soon take place, if not, the development of evidences of pleurisy with effusion, which may become purulent, soon follow.

The physical conditions suggest percussion as a primary method of diagnosis.

Percussion.—Naturally you assume that the sound must be tympanitic or hollow. The pitch is lower than the tympany of most cavities—it is lower than the pitch of the stomach tympany. The reason is obvious: the cavity is very large, the vibrations of the pulses of air are long, yet the walls are tense. It is for this latter reason, especially, that the quality of the sound is tympanitic. At times, although the cavity may be large, the lung may be bound down by adhesions, and the orifice of communication between the cavity and the lung may be closed; the tone, in these circumstances, may be so high pitched, that its quality may be imperfectly developed. This fact, and the possibly associated conditions of thickened pleura, or thick layers of external tissues, render the sound at times a little dull. Tympanitic dullness is the term best adapted for these cases.

Patulous Pulmonary Fistulæ.—If auscultation is practiced over a large air-containing cavity, while an assistant strikes the surface of the chest by means of two coins (silver dollars), a beautifully clear, ringing sound will be heard, in tone not unlike the stroke of a bell of a small clock. This sound is not heard unless intra- or extra-pulmonary fistula exist.

Auscultation.—The paradoxical expression, to hear silence, is the cardinal evidence of pneumothorax. More truly can this be said than in the similar separation of the lung from the chest wall, which occurs in pleurisy. The compression exerted by the air upon the lung is so great, that all transmission of the respiratory murmur is abolished. Of course, the compressed lung is to be detected by bronchial breathing at some point, usually near the spinal column. In some cases, the conditions surrounding the perforation in the visceral pleura may keep it more patulous than in others. In this case, the air rushing into the artificial cavity formed by the separated pleural surfaces, the respiration obtains an amphoric cavernous tone. If drops of fluid escape from the lung, and fall into the cavity, or if bubbles of fluid are generated on the surface of the pleural effusion, in these cases, the sounds are often echoed to the ear with a metallic ring, resembling the tinkle of a silver bell.*

The presence of this sound signifies the patulous state of the pulmonary fistula, its absence does not necessarily mean that the opening is closed permanently by inflammation, since the closure may have been effected by the other causes, such as partial inflation of the lung, position, and the like.

The results of *succussion* are a splashing sound when the patient is abruptly shaken. The sound being not only audible, but can be detected as a fremitus.

Inspection.—The ingress and egress of the air into the pleural cavity widens the chest, and the intercostal spaces also become effaced. In other respects the remarks on the subject of unilateral enlargements, in the chapter on unilateral pleurisy by inspection and mensuration, are apropos.

The chest is more motionless than in pleural effusion, though some up and down movement of the ribs may occur. Displacements of the intra-thoracic viscera occur, as in pleurisies with effusion.

Vocal resonance and fremitus must be negative. The recognition of the fluid in the base of the chest is dependent on the ordinary signs present in uncomplicated pleural effusions. This is illustrated by the transformation of a case of hydro-pneumothorax into one of chronic pleurisy with effusion—the most favorable termination of pneumothorax.

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* A large cavity may yield this tinkle, or some liquid in a large bronchial tube.

Differential Diagnosis.—Bilateral emphysema is frequent. Pneumothorax is almost always unilateral. Cases of bilateral pneumothorax have been cited, but they are so unusual as to be among the curiosities of medical literature.

From Unilateral Emphysema (vicarious).—Remark, in pneumothorax, the nearly suppressed or absent respiratory murmur, and the existence of the metallic tinkle. Succussion, too, is available. The respiratory movements are also more suppressed in pneumothorax, and the displacement of the viscera different and more marked than in emphysema. The percussion pitch is lower than the tone, in emphysema. Although both notes are tympanitic in pneumothorax, there is a less vesicular quality. Finally, the definite antecedents of emphysema differ widely from pneumothorax, which is suddenly developed.

A distended stomach may become a source of error. If some water is sipped, the tinkle following its entrance into the stomach is very audible.

Large cavities at the base might mislead, particularly if the pneumothorax is encysted.

In the first place, if we nucleate the facts gathered from the clinical histories, we find that in cases of large cavities, in addition to the evident signs of wasting; bilateral disease; respiration is also audible throughout a large area of super-imposed lung. The encysted variety of pneumothorax is very rare. We may find the tone of the tympany is lower than in cavities. The prominent interspaces and local distention of the chest, and the possible evidences of liquid effusion—all may help to decide obscure cases in favor of pneumothorax.

CHAPTER XIII.

MALIGNANT DISEASE OF THE MEDIASTINUM.

We would wish to nucleate a few of the facts by which malignant disease of the mediastinum or lungs may be recognized. To describe all the physical signs contingent on these growths, would be almost endless labor. There are two forms of disease, the primary and the secondary. The history, both personal and hereditary, should be carefully analyzed. In one case, which we recall, of apparent primary cancer of the lung, the diagnosis was finally decided in favor of malignant disease, because fifteen years before the patient had undergone an amputation of the leg for "some tumor." In malignant disease, pleural effusion is a frequent concomitant. If it occur in connection with mediastinal tumor, it may arise through pressure upon the azygos or hemi-azygos veins, thus preventing a free return of blood from the pleural veins, with resultant hydrothorax. The effusion, however, may be turbid, highly albuminous, with a large proportion of coagulable fibrine, the result of inflammatory process, the sequel to malignant disease, developed secondarily in the pleura. The presence of a nodule or nodules of malignant nature in the lungs, when large enough to be appreciated by physical diagnosis, usually yield a very dull or flat note in percussion. The respiratory murmur is some phase of bronchial breathing, or if a bronchus is pressed on, may be feeble or suppressed over the affected side. Over the bronchial tree, behind the third dorsal vertebra, the breathing is feeble or whistling if the primary bronchi are compressed. As for expectoration, if bronchitis exists the sputa may be purulent; if the new formation undergoes softening, possibly bloody, resembling prune juice or black currant jelly. The cells constituting the growth, with portions of pulmonary structure, may be found on microscopic examination of the sputa. Primary cancer of the lungs, or that propagated from the mediastinum, is usually unilateral; if secondary to disease elsewhere, it may be bilateral. Inspection may show retraction or expansion of the chest, necessarily the latter, if pleural effusion exist.

In cases of pulmonary cancer, if we examine the supraclavicular regions by palpation, we can often discover masses of painless, movable glandular enlargements equaling the size of hickory nuts. The nodules in the lungs vary from the size of a chestnut to a lemon, or diffused cancerous infiltration may be disposed throughout the various lobes. Cough, pain or dyspnœa may develop the impression that phthisis co-exists. Even pyrexia may become a symptom, with night sweats confined to the *affected side* (*Walsh*). Still the elevation of temperature is less than in phthisis. Phthisis is a rare coincidence of the history of malignant disease, and occurs mostly at an earlier period of life. Walsh asserts the disease has a mean duration of 13.2 months, a minimum of 3.5 months, the maximum at 27 months; but he acknowledges that this is based on a small contingent of cases.

Bronchitis, with atrophic emphysema, simple pleural effusion, chronic pleurisy with retraction, fibroid phthisis, are the diseases with which it is most likely to confuse a case of cancerous disease.

The characteristic history of these diseases, elsewhere detailed, the cancerous hue of the skin, and the history of the case, are helpful evidences on the side of malignant tumor. Mediastinal tumors must be distinguished from aneurism. Some cases of intra-thoracic tumor involve all the acumen and research of the most accomplished diagnostician. In speaking of tumors of the mediastinal region, an interesting condition is that of enlargement of the bronchial glands. It is a process which occurs in scrofulous persons in some cases of early syphilis, and which has been noted as an incident of the history of various forms of respiratory disease. It is associated with some forms of pertussis, rachitis, and as a possible incident of various pulmonary catarrhs. The physical signs by percussion, as demonstrated by M. Guineau de Mussy, consist in percussion over the spinous processes of the cervical vertebræ, the course of the trachea. Following this line in the healthy subject, a distinct tubular sound is elicited by percussion down to the point of bifurcation of the trachea at the level of the fourth dorsal vertebra. Opposite the fifth and downward we get the lower-pitched pulmonary resonance. When the tracheal and bronchial glands are enlarged, the tubular sound over the upper dorsal vertebræ is replaced by dullness, which may contrast sharply above with the tracheal, and below with the vesicular resonance.*

The respiratory murmur will be feeble on one or both sides of the chest, especially over this same interscapular region. Over one or the other bronchus, the respiratory murmur may be high pitched, more marked than in health.

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* A contribution to the History of Influenza, by Drs. White and Guiteras, Phila. *Med. Times*, April 10, 1880. It is also associated with some cases of aneurism. *See* Path. Trans., vol. 17, London.

For much interesting matter and references to lesions connected with enlargement of the bronchial glands, we refer those interested to this paper. The writer has seen cases of enlargement of the bronchial glands in syphilis, and in scrofulous persons. The enlargement, if serious, is capable of originating many symptoms of intra-thoracic pressure—such as hoarseness, almost amounting to aphonia, dysphagia, unequal pupils, a sense of oppression at the root of the neck, dyspnœa, etc. It is also capable of exciting serious mischief in the lungs—such as atelectasis, emphysema, and certain forms of pneumonia.

CHAPTER XIV.

RESPIRATORY PERCUSSION.

Respiratory percussion has already been described, as it can be practiced on the normal chest. It remains for us to indicate its value in the examinations made in disease.

Manifestly the method depends on the pervious state of the air passages and the vesicles, and upon the principle of physical diagnosis already laid down. Each observer will perhaps extend the scope of the method for himself. The writer has practiced the percussion in the following conditions, and unites with Dr. Da Costa in his conclusions: "In marked emphysema the excessively clear vesiculo-tympanitic note is unchanged by percussion during the act of breathing; when the emphysema is not so great, it is but slightly changed. *But if emphysema be present at all, except to a trifling degree, the sound is not much altered.*" Since emphysema, with attendant enlarged and dilated right ventricle, is one of the most serious complications of asthma and of chronic bronchitis, how valuable it becomes to add this corroborative test. In pleurisy, full inflation will strikingly bring out the *abrupt contrast* between the pulmonary resonance above and the flat note below. When pleurisy co-exists with pneumonia, and there is blowing breathing at the back of the lung, this method is very helpful. If there be pneumonic consolidation at lower part of the chest, the flat note remains unchanged; and so will the dullness of the upper part be unchanged by forced respiration. But if the tubular breathing be simply from compression, or condensation of the lung, and not from hepatization, decided clearness takes the place of the dullness."

"At the base of the chest, if there be chronic pneumonia, the line of dullness changes in part, and if there is no sharp line of contrast between dullness and resonance developed on full breathing, it is consolidated lung. When, by respiratory percussion, the dullness at its uppermost limit becomes sharply defined, while it is unaltered below, it is an effusion." "In bronchitis the percussion resonance is, practically speaking, unaffected. Yet where extremely abundant secretions exist, and obscure breath-sounds, the clearness of note may become impaired, and we are in doubt as to the state of the pulmonary textures. Respiratory percussion removes the doubt; the chest struck while in a full respiration returns a sound exactly corresponding to the sound we should obtain in health. If, however, there be an extension to the finer structures, and beginning consolidation, the note does not become fuller and more resonant, and the difference between the damaged point and the surrounding parts, or corresponding portions of the other side, is being manifest. If, however, the lung be merely collapsed, respiratory percussion gives an almost normal sound, unless the collapse be extensive, and the power of expanding the lung be lost, or inflammation beset the collapsed lobules." "In acute lobar pneumonia, as resolution begins, the note heard on respiratory percussion is more resonant, more pulmonary. This change may show itself in advance of the râles redux."

"In heart disease there is often congestion so pronounced as to lead to apprehensions of consolidations; these are removed when forced inspiration clears up the suspected points."

"There is a single point linked with phthisis, which is useful in connection with what has been said in connection with feeble breathing, in cases of phthisis. When in a case of phthisis we find that the dullness on percussion is no longer modified by fixed inspiration, we have a certain test of the malady having progressed."

In pneumothorax the doctor thinks that if inspiration does not change the percussion note, the opening is closed. If the tympanitic or amphoric note is changed essentially, we may, he thinks, infer that the air still rushes from the lung into the artificial cavity in the pleura.

These quotations, reproduced in the distinguished author's language, are well calculated to arrest the attention of all thoughtful non-routine students.

"In regard to the cavities, the pitch of the sound is raised so much that the quality sometimes appears dull, or that intermediate type, tympanitic dullness, may prevail."*

This method of diagnosis is especially useful in children as a means of differentiating between capillary bronchitis and catarrhal pneumonia; the best opportunity is when the child gasps in the act of crying.

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* See *American Journal of Medical Sciences*, for July 1875.

PART II.

DISEASES OF THE HEART AND PERICARDIUM.

CHAPTER I.

PROCESSES WHICH DEVELOP CARDIAC DISEASES.

The symptoms of cardiac disease are protean, partly because the vital functions of the entire economy depend upon the efficiency with which the circulation is carried on; partly because cardiac disease is entailed as the result of many various morbid physiological processes. In the study of the lesions of the heart and arterial system, these isolated phenomena must be defined and traced to their source; then the results of physical diagnosis must be nucleated with the above study. Neglect of this method is a fruitful cause of many of the failures in the correct appreciation of the bearing of physical diagnosis on the therapeusis of heart disease. Physical diagnosis of the heart employs the senses of sight, touch, and hearing; but we propose, as an initial step, a glance at the various factors which originate heart disease. Second, the diagnosis of valvular lesions of the heart. Thirdly, we will study the local effects of valvular cardiac disease upon the cardiac substance, and afterward the symptomatology of the lesions developed in the general system, as the outcome of valvular disease.

We shall endeavor to blend the study of valvular disease with this study of symptomatology, because the separate recognition of a valvular disease is useless without a correct appreciation of the consequences of the lesion. Necessarily limited space will compel us to deal only with the outlines of pathological processes, and to dwell only on those bearing near relation to the diagnosis.

In the first place, rheumatic fever is delineated with striking boldness as a fundamental antecedent of a large proportion of lesions of the cardiac system. In Dr. Sansom's writings on this subject he says, "that taking seventy-seven cases in which a precise early history could be obtained, thirty-four occurred in those who had suffered one or more attacks of undoubted rheumatic fever; in thirteen rheumatic pains, not sufficient to keep the patient in the house, had been noted. In fifteen there was no history of any rheumatic affection whatever; if any symptoms were mentioned they were merely those of indigestion. Rheumatic gout had been the portion of two patients, scarlet fever of three, and typhoid, or low fever of four. In six cases the evidence pointed to the conclusion that the disease was congenital."

The causes of heart disease may be grouped according to the portion of the cardiac system upon which they are usually expended.

Rheumatism and its allied states are cardinal causes of endo-cardiac change with valvular lession.

Typhoid and the zymotic fevers all weaken the heart substance by impairing the constitution of the muscular tissue, and dilatation is threatened.

Alcoholic indulgence, syphilis, imperfect or improper nutrition, can result in similar changes, but also affect the arterial system. The puerperal state sometimes induces hypertrophy; muscular strain, over exercise, etc., also tend to produce hypertrophy. In all lesions of the walls, mechanical principles give direction to the changes; this is especially true of changes observed in valvular lesions. A study of the condition of the muscular tissue of the heart is of vast importance; in fact, without the appreciation of this point, a study of valvular disease is nearly barren of useful therapeutic hints. We must recognize that hypertrophy is associated or terminates in dilatation, which means cardiac weakness. In continuing our summary we find that processes which involve structures contiguous to the heart are potential in the etiology of the disease of the heart and pericardial tissue. For instance, various diseases of the lungs result in venous repletion and dilated hypertrophy of the right heart, while pleural disease may be the parent of pericardial lesion.

Deficient assimilation or elimination find an expression in increased arterial tension, and sow the seeds of arterial and structural, or functional cardiac diseases. Bright's diseases frequently induce change in the cardiac substance (hypertrophy), or in the endo- or pericardium. But the influence of renal disease deserves a special consideration. Renal lesions not only develop cardiac disease, but occur as a sequence. When structural disease affects the kidney, elimination of the urinary solids is imperfect, their retention results in vaso-motor irritation and contraction.

In long continued irritation the effect on the heart is similar to that which prevails in any over-taxed muscle, viz., hypertrophy, because the onward transit of the blood in the arterioles depends on the increased cardiac power. The hypertrophy is chiefly limited to the left ventricle; if, however, renal disease attack a patient already reduced in strength, with impoverished blood tissue (as in phthisis), hypertrophy cannot be invariably looked for. In the more rapid phases of renal disease the increase of vaso-motor tonus is not sufficiently long continued to result in hypertrophy.

Renal disease occurs as a sequence to cardiac disease, because the kidneys, like the other viscera, suffer venous repletion, and if this be long continued, a form of mingled tubular degeneration and intra-tubular growth may result in the so-called cardiac kidney. The evidence of these changes is quickly seen in albuminuria, by dropsy commencing in the feet, finally by hyaline tube casts. A special feature of cardiac dropsy is the absence of the white skin and anæmia of renal dropsy; instead, we notice the cyanosed skin of venous congestion.

In the gouty state there is always a tendency to increased vaso-motor tonus, and atheroma; both conditions favor hypertrophy, since the aortic elasticity is gradually abolished.

CHAPTER II.

DIAGNOSIS OF VALVULAR DISEASE. ASSOCIATE SYMPTOMS AND CHANGES IN THE CARDIAC SUBSTANCE, INCIDENT TO VALVULAR DISEASE. "MITRAL AND TRICUSPID LESIONS."

We are now prepared to study the diagnosis and the local effects of valvular lesions upon the cardiac substance. With each form of valvular disease we shall try to outline the symptomatology of the lesions developed in the general system as the result of cardiac incompetency.

One of the most valuable methods of investigation is auscultation, and we shall study first the evidences of valvular disease by auscultation.

Auscultation.—We employ two terms in the physical diagnosis of the heart, sounds and murmurs. To the first we assign the representation of the audible sounds which occur during the normal revolution of the heart. The word murmur is reserved to indicate the adventitious sounds induced by lesions in the valves, or modifications in the blood tissue. In common with the physiologists the sounds of the heart are represented to the diagnostician by the terms first and second sounds. Several elements are combined to produce the first sound: it is partly the result of closure of the valves of the mitral and tricuspid orifices; partly the whirl of the blood within the cavities of the ventricles; partly the sound produced by the contraction of the muscular walls of the ventricles. This first sound, called systolic, represents the period during which the blood is projected from the left ventricle into the aorta, and from the right ventricle into the pulmonary artery. The interval between the systolic or first sound, and the so-called second or diastolic sound, comprises the short pause. The diastole of the heart comprehends the time during which the blood is flowing through the pulmonary artery and the lungs, thence via the pulmonary vein into the left auricle, thence into the left ventricle again, also the time occupied by the transit of the blood through the systemic circulation back to the right auricle and right ventricle. The diastole, then, is a period marked at its commencement by the closure of the aortic semilunar valves and the like closure of the valves of the pulmonary artery. The click connected with the closure of these valves is the accredited cause of the second sound. The interval following the closure of the valves constitutes the long pause, which is terminated by the contraction of the two auricles, by which they are thoroughly emptied of blood. The sound produced by the auricular systole is inaudible under physiological conditions, but the fact is important to a clear estimation of the murmurs developed by mitral obstruction.*

Let us describe the sounds more minutely. The first sound is a long, low-pitched, muffled sound, in sharp contrast with the high-pitched, sharp, and clacking second sound. It will be found that in disease both these sounds become radically altered. The first sound, losing the muscular element, becomes less muffled, and as clacking or valvular as the second sound, or the sound is entirely lost. The attributes of the second sound sometimes become very markedly intensified, meriting the term accentuated, or the sound may become very feeble. We now must ascertain the localities at which the sounds are best heard. It is an axiom, that both sounds and murmurs are most distinctly heard at that portion of the chest surface at which the cavity in which the sound or murmur is generated approaches most closely the chest surface. Now, the left ventricle is most superficial around the region of the apex beat, the aorta at the junction of the second and third ribs with the sternum.

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* The cardiac revolution is represented by the illustration at end of volume.

The right ventricle is most superficial near the ensiform cartilage; the pulmonary artery crosses in front of the aorta, and is most superficial near the junctions of the second and third ribs with the sternum; but we practically estimate the first and second sound as though they were produced in the left side of the heart alone. In valvular disease, we oftenest discover murmur on the *left* side only, but many concomitant modifications of the sounds occur on the right side. We commence our study of a sound at the base, because we know that the valves are clustered at that point; we then proceed either downward to the apex, or upward to the second right costal cartilages. With a knowledge of the method of the production of a cardiac sound, and its distribution, let us next inquire how we differentiate the one sound from the other.

We have at command the following tests:—

1st. Sound.—The quality is muffled, long, low-pitched, phonetically represented by the word lupp. It is best heard at the apex beat. It is synchronous, not only with the apex beat, but also with the carotid pulse. It just precedes the radial pulse by a hardly noticeable interval.

2d Sound.—The quality is clear, clacking, or snapping, and high-pitched, phonetically represented by the word dupp. *It succeeds the apex beat* and cardiac pulse. It is best heard at the second right costal cartilage, commonly called the aortic cartilage.

While all this is true, one must depend mostly upon the time, since diseased states modify the quality of the sounds, and at all times one can hear both sounds all over the præcordia, so that sometimes it is impossible to define the situation of maximum intensity. But invariably the sound which occurs with the systole of the ventricle is the first sound, the one following the apex beat is the second sound. It is desirable, as a secondary process, to time the sounds, and estimate their quality at the ensiform cartilage over the right ventricle, and also over the pulmonary or second left costal cartilage.

It is next in order to consider the lesions of the valvular elements of the cardiac structure. Space will not permit a detailed description of their character. We must simply say, that the results of inflammation can alter each valve, so that its orifice on the one hand is roughened or contracted, on the other hand the valve may be nearly destroyed, it may be thickened, fringed with vegetations, or adhesions of the columnæ carneæ may bind down one or more leaflets. The result in each of these conditions is functional incompetency of the valve. In the mitral or tricuspid valves a clot may become so entangled in the columnæ carneæ, or chorda tendineæ, that insufficiency, or a murmur simulating insufficiency, may be produced. (*See* Thrombosis.) The vegetations and other products of inflammation may undergo calcareous degeneration, or this form of degeneration may be the result of atheroma induced by the arterial overstrain, or the degeneration consequent on advancing years. For purposes of clinical study, let us nucleate these facts by the statement that valvular lesions result in narrowing or stenosis, or in roughening, or in such contractions that the valves become insufficient. Any of the valves, theoretically speaking, can undergo these changes.

Practically, primary lesions are confined to the valves of the *left* heart, although lesions of the right heart are not infrequent as secondary to left-sided disease.

We have commonly five murmurs represented for our study: three of insufficiency, with regurgitation; two of obstruction, or stenosis.

Tricuspid valves	Obstruction very rare.	
	Regurgitation.	Nearly always secondary. Time systolic.
Pulmonary artery Semilunar valves	Obstruction Regurgitation	Unknown unless congenital.
Mitral valves obstruction		Post-diastolic, presystolic. Mitral direct. Auriculo systolic—occurs at end of diastolic period.
Regurgitation	Systolic.	
Aortic valves regurgitation	Diastolic at commencement of diastolic period.	
Obstruction	Systolic.	

In the study of murmurs there are some points of importance to which allusion has not yet been made. In the first place, the quality and pitch of murmurs indicates absolutely nothing as to the extent of the lesion.

A most insignificant lesion may be manifested by a most portentous murmur, while a most serious lesion may give rise to a most insignificantly loud murmur. The single method of estimating the gravity of murmurs indicative of valvular lesion, is by noticing the degree of change in the cavity in which the murmur is generated, and estimation of the degree of diminution of cardiac propulsive power, as manifested by a failing circulation.

As a rule, *aortic* murmurs develop in persons of middle and advanced life, in consequence of slowly induced inflammatory change developed by continuous overstrain, the result of a high arterial tension. These changes occur in those subject to chronic renal disease, to syphilis, to laborious avocations, to indulgence in alcohol, to the gouty state; or the murmurs may be the result of calcareous atheroma incident to degenerations of advancing years. This is especially true of aortic regurgitation. On the other hand, *mitral* disease is more directly the most serious complication of acute rheumatism, or the rheumatic diathesis. The rule is, however, not unvarying. Young persons suffer from aortic disease, particularly aortic obstruction, and old persons frequently present a long-standing mitral lesion.

Another point of interest relates to the pitch of murmurs. Mitral murmurs are usually low-pitched, blowing sounds; aortic murmurs are usually high-pitched, cooing or grating sounds; but the rule is by no means unvarying.

Finally, a murmur may replace entirely the normal sound, or some portion of the normal sound can still be heard. In the latter case, the destruction of a valve is not so complete. Degenerations of the heart and weakness of cardiac muscular power can sometimes abolish a murmur previously distinct, the murmur reappearing if the cardiac power increases under treatment. The murmurs resulting from acute rheumatic endocarditis may vanish in time, as the swelling subsides, and the edges of the valve again approximate normally. More commonly the leak increases in direct relation with the lapse of time, and the tension maintained within the ventricle by the demands of life.

If care is taken to conserve a minimum degree of intra-ventricular tension, by constant supervision (especially of the habits of life), the rapidity of the progress of the lesion is much reduced. Care must also be observed to prevent a repetition of attacks of acute rheumatism, each of which tends to aggravate the mischief. We have said the murmurs, acoustically speaking, are soft and blowing. This is particularly the case with recent murmurs, but if spiculæ of calcareous degeneration project into the circulation, the murmur may be roughened, and may be characterized by some appropriate adjective. All cardiac murmurs, like the cardiac sounds, can be heard at the base, and follow the law which prescribes their location of maximum intensity to that situation of the chest at which the cavity in which they are produced approaches *most directly the surface of the chest. They are subject to another general law, viz., that they are propagated in the direction of the blood current by which they are developed.*

Mitral Regurgitation.—Having heard the murmur at base, proceed to auscult the aortic cartilage, and then the apex. If the murmur be generated by regurgitation through the mitral valve, the murmur will be loudest at the apex. The next step will be to time the murmur, which can be done, by noting the *synchronous beat of* *the apex*, or the *carotid artery*; in other words, time it as you would the first sound. Often, in commencing lessions of the mitral valve in acute rheumatism, the murmur will not be transmitted outside the apex; but if there is much regurgitation, the murmur will be transmitted in the direction of the blood current, *e.g.*, back into the auricle, through the pulmonary vein into the lungs. The murmur can be traced to two sites on the chest, one the axillæ (since the tissues covering the chest wall are thinnest at this point), and the other at the angle of the left scapula and the vertebral column. In the axillæ, always listen above the line of the apex beat; a recent murmur can often be heard at this point that is not transmitted as far as the scapulæ. Sometimes the amount of swelling is sufficient to thicken the first sound, or develop a slight murmur, heard at the apex and for a short distance outside; in this case the amount of regurgitation is slight, and if the lesion occur in a case of acute rheumatism, the murmur may disappear after a time. On the other hand, a moderate lesion, if associated with much calcareous degeneration, may give origin to a murmur which may be transmitted to a great distance. The size of the orifice also gives expression to the intensity of a murmur. All that has been said with reference to a murmur refers to the line of transmission with *maximum* intensity. The murmur of mitral regurgitation may be heard at the ensiform or the aortic cartilages, or over the auricle, but is evidently not so pronounced as in the line of the blood current, by which the murmur is developed.

Exceptions.—Mitral regurgitation may exist without murmur in some cases of extreme dilatation, in which case the heart muscle is so weak that so little blood regurgitates through the mitral valve that murmur is not developed. In these cases, also, a murmur may be heard in the erect but not in the recumbent posture, or contrariwise, and can be explained in the same fashion. It is quite possible to auscult the heart without recognizing a murmur, which develops as the heart systole improves under rest and treatment. In these cases all the evidences of venous congestion prevail. The diagnosis of a mitral murmur is incomplete unless the study is interlaced with a consideration of the general and local consequences of valvular cardiac disease. The evidences of the gravity of the lesion are to be drawn from the effects upon the cardiac substance and from the signs of failing circulation in the general system.

The local effects of valvular disease find their primary expression upon that cavity of the four which is most directly affected by the abnormal circulation; afterward the adjacent cardiac cavities become more or less involved. The first demand of mitral insufficiency is, that increase of propulsive power by ventricular systole is indispensable. Hypertrophy is the primary response, but in most cases hypertrophy is inadequate to remedy the lesion permanently, or even for any considerable period. This arises, partly, from the fact that hypertrophy tends to force the blood in jets through the abnormally patulous orifice, at the same time that it increases the force of the current into the aorta. The largest amount of blood naturally passes into the aorta, since the aortic orifice is the larger orifice—and for a time the hypertrophy is compensatory. But all the while the increased power of the systole tends to increase, little by little, the regurgitation and widen the abnormal valvular orifice. It soon transpires that the aorta is inadequately filled—a condition fruitful of evil. The coronary arteries are imperfectly filled by the imperfect aortic systole; dilatation, with fatty degeneration, speedily ensues.* How rapidly, will depend on the degree of mitral lesion.

The left auricle, in cases of long-standing lesion, becomes hypertrophied and dilated; next ensues repletion of the pulmonary vein, with subsequent congestion of the lungs, with hypertrophy, and dilatation of the right ventricle. Enlargement of the right auricle occurs in its turn, with a more or less pronounced repletion of the entire venous system throughout the body. The results of mitral disease upon the system at large include congestion of the liver and kidneys as the most important lesion in the chain. The liver is enlarged, and may pulsate synchronously with the systole. Mesenteric congestion, with symptomatic indigestion from imperfect glandular functional activity, tympanites, ascites, hemorrhoids, are all incidents of this sketch. The renal congestion manifests itself by albuminuria, followed by anasarca, which commences in the feet and legs; the reverse is true of initial kidney dropsy, which begins in the cellular facial tissues. General anasarca is succeeded by effusions into the serous sacs, ascites, hydrothorax. In the latter case it is either bilateral, or, if unilateral, it is disposed upon the left side of the chest, perhaps, because the enlarged heart presses upon the internal thoracic veins on the left side. Since both hepatic and renal congestion is evidenced by symptoms — query:

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* *See* article on Dilatation with Hypertrophy.

what are the symptoms of pulmonary engorgement? The reply is, intercurrent attacks of hæmoptysis; moreover, the pulmonary congestion is a fruitful harbinger of catarrhs, excited by atmospheric influences; so we add to the list bronchitis, more or less grave, as a frequent complication. Later in the case œdema occurs, as little by little the hepatic and renal activity is reduced, and the circulation is stored with imperfectly elaborated or eliminated nitrogenous materials. The sufferer is consequently liable to any form of serous inflammation, and finally to a possible death from uræmia. Death, it is true, may suddenly terminate the scene, as the result of cardiac failure, but more frequently the patient succumbs to some of the incidental complications. The grave symptoms linked with mitral disease indicate that its duration must be largely measured by the number of complications, and the rapidity of their advent.

The procession of complications are influenced by the avocations of the patient, the presence or absence of syphilitic poisoning, the alcoholic habit, or the recurrence of endocarditis. The severity of the lesion, of course, has a primary and potential influence. Age is also a factor of peculiar import. Even serious mitral lesions in the very young are modified and sometimes vanish as maturity approaches.

Tricuspid regurgitation is a secondary process, in almost every case, to continued pulmonary congestion, which results in hypertrophy and dilatation of the right ventricle. The repletion of the right heart is due to pulmonary conditions, notably emphysema, or else to left sided valvular disease. The lesion is of two sorts first, the continued over-distention leads to hypertrophy and thickening of the chordæ tendineæ, and the leaflets themselves. Incompetency and regurgitation with attendant murmur may follow. Or, without valvular change, simply as the result of dilatation of the right ventricle, the leaflets of the tricuspid valve, which are normally competent to close the auriculo-ventricular aperture during systole, are withdrawn from one another by circumferential traction of the walls of the widened ventricle, so that their edges do not perfectly meet. In systole, therefore, blood regurgitates into the auricle. Lesions have been recorded of inflammation of the right ventricle, but they are rare.

With the above ideas in mind, it is easy to understand that the tricuspid murmur is often temporary. It may be absent, owing to extreme dilatation of the walls of the ventricle and their consequent weakness. The murmurs can be developed or increased by exercise, which favors congestion of the lungs; it will sometimes disappear if a state of rest is maintained. The murmur may be confused on the one hand with a mitral systolic; on the other, with an aortic obstructive, or systolic murmur; the safest guide will be observation of the associated symptomatology. We are also to consider the difference in pitch of murmurs possessing the same time. In mitral systolic murmurs we have a different position of maximum intensity and area of distribution. With aortic systolic murmur in addition to the above points, there is always a dilated, or hypertrophied left ventricle.

The area of *transmission* of a murmur of tricuspid regurgitation is in harmony with the rule. It is heard at the base, it is transmitted downward, a little to the *right* of the sternum, with maximum intensity, because the right ventricle, if enlarged, always extends to the right of the sternum and is most superficial just to the right of the ensiform cartilage. It is often heard over the body of the ventricle, but is not loud enough to be carried to the right axilla; indeed, tricuspid murmurs are usually very soft, and blowing in quality. When the murmur is present we usually have distention of the veins of the neck, more especially of the *jugular* external and internal. In this connection, a word or two on venous distention is pertinent.

"In healthy persons the internal jugular vein is not visible; the external jugular, on the contrary, is visible in the erect, if not in the recumbent, posture of the body. The external jugular vein usually possesses two sets of valves, one at its mouth and one in the middle of its course. The internal jugular is provided with valves at or a little above its mouth. All these valves are very variable, both as to number and position. Moreover, they are often quite incompetent to close the vessel. This is especially the case with the valves in the internal jugular. The right internal jugular vein, right innominate vein and vena cava superior, form a continuous channel, which is almost straight. For this reason all the signs about to be described are more marked on the right side of the neck than on the left, and they would be always more marked in the internal than the external jugular were it not that the deep position of the former vein is a hindrance to observation."*

Tricuspid disease predisposes to auricular fullness, and, in turn, to distention of the vena cavæ and the jugular veins. Naturally the overfilling is not absolutely permanent, but dependent on the causes which give expression to the tricuspid murmur. There will usually be a venous pulse, direct or indirect. Indirect may be due to the fact that an over-distended ventricle bulges the tricuspid valves toward the auricle, and gives origin to impulse with each systole, which is measurably counteracted by the auricular systole. Direct pulsation occurs in consequence of a true regurgitation and propulsion of blood into the jugular veins through incompetent venous valves.

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* Gee, Physical Diagnosis.

Gee says that we can distinguish whether the valves are competent or not by compressing the veins in the upper part of the neck, and observing whether they are filled with blood from below. The rise of blood and its pulsation from below is made characteristic by this method. The pulsations of the carotid artery may communicate systolic pulsation to the veins. The practice just mentioned, and a condition of the heart predisposing to distention of the right ventricle, are the best safeguards from error. The jugular veins are, of course, liable to overfilling from other causes, such as pressure by pleural or pericardial adhesions, thrombosis, etc., acting on the vena cava or innominate. A permanent or long-continued repletion of the veins occasions marked dilatation, most commonly of the right side, sometimes upon the left. Venous distention, which has a pulmonary origin, is possible. The descent of the diaphragm in normal inspiration is one of the motive powers of the circulation. In diseases in which this action of the diaphragm is interfered with, as emphysema, with chronic catarrh, the veins are always more or less distended, as a result of the pulmonary obstruction. From these pulmonary conditions distention of the right ventricle may occur, demonstrated by the abnormal boundaries assigned to it by *percussion, inspection* and palpation.

CHAPTER III.

MITRAL OBSTRUCTION.

Mitral Obstruction.—The auriculo-ventricular valvular orifice may also be roughened or obstructed. The form of obstruction is so uniform that it has suggested the possibility that the lesion is congenital, rather than the result of endocarditis. The question is plausible enough to lead one carefully to study the history of each case of mitral obstruction. There is no doubt, however, that, in common with other lesions, it can arise from endocarditis.

The shape of the mitral orifice merits passing notice, in addition to what has been already stated in reference to valvular disease in general. The average circumference of the normal orifice is about four inches; the form is oval, with correspondingly narrow-long diameter. In disease, this orifice is seriously modified. The chorda tendineæ and the valve may be thickened, stiffened into a rigid mass, or the orifice may be obstructed by vegetations. The leaflets may be fused so as to form a more or less conical tube, its smaller extremity opening into the ventricle; sometimes the orifice is extremely small, admitting only the little finger, or even a pen handle through it. The orifice may be slit-like or "button-hole" in appearance. The valve may be encrusted with calcareous salts, of bony hardness, in the same way as other forms of valvular disease. The effect on the auriculo-ventricular aperture is, of course, opposite to that of regurgitation. The orifice is narrowed, not widened. Instead of narrowing—or stenosis—the *auricular* surface of the mitral valve may be simply roughened by vegetations, without materially obstructing the orifice. Presystolic murmur is associated with both conditions, but is best developed if stenosis exists.

In reference to the murmur, the time or the rhythm in which it occurs has been variously designated by the terms, presystolic, auriculo-systolic, post-disatolic, or simple initial obstructive murmur. (*See* Illustration.) The murmur may include the entire disastolic period, if the obstruction is extreme; on the other hand, it may only occur at the end of the diastole, immediately before the systole, thus meriting two of the terms, post-diastolic or presystolic. Custom has applied the term diastolic to the murmur of aortic regurgitation; in reality this murmur occurs at the commencement of the diastole, so the best term for the murmur of mitral obstruction or roughening is presystolic. The facts are, that at the end of the diastolic period both auricles contract, to express from their cavities the last remnants of blood and complete the filling of the ventricles. If mitral obstruction or roughening exist, it is evident that the murmur thereby generated will occur just before the systole, before the impulse of apex or carotid, after the second sound. The murmur has in most cases *two* centres of equal intensity. The one corresponding with the auricle, the other with the apex, to which it is carried by the blood current. It must be remembered that at the time the murmur is produced the apex is not applied to the chest wall until the systole occurs. Perhaps this explains the fact that the murmur is best heard a little within the line of the apex beat; whereas in mitral regurgitation with systolic murmur, the sound is conducted to the ear when the ventricle is in approximation with the chest wall. A mitral murmur may sometimes be heard just outside the apex, because the ventricle may be both hypertrophied and dilated. Presystolic murmur is localized, and cannot be transmitted from the apex, because the blood current is immediately diverted into the aorta by the ventricular systole. Thus it happens that the murmur is, as it were, clipped or cut off short at the apex. In regard to the cases in which there is a murmur with a centre of maximum intensity over the auricle, we must remember that in health the left auricle is placed beneath the right, so that in cases of simple roughening of the auricular surface of the mitral, the murmur may not be loudly heard over this cavity. But if true stenosis exist, one of the earliest consequences will be the enlargement, by hypertrophy and dilatation, of the left auricle, and the murmur will then be heard over the area included by the auricle.

Concerning the *exceptional* transmission of presystolic murmurs into the axillæ, or posteriorly, as reported by Dr. Andrews,* we recognize that such transmission is possible, but an unlikely event. In his cases there may have been pulmonary consolidation, or pleural adhesions, or the murmurs may have been exceptionally loud.

Dilatation and hypertrophy may become so extreme, owing to long continued and serious obstruction, that the normal præcordial area may be filled by the dilated auricle, the left ventricle may be correspondingly depressed and displaced. In some cases, the enlarged auricle presents a visible tumor, causing bulging of more or less of the præcordial area.

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* St. Bartholomew's Hospital Report, 1877.

The auricle may become so much distended as to admit two good-sized Sicily oranges into its cavity. The hypertrophy and dilatation exceed the similar condition in mitral regurgitation.*

In natural sequence we often find very marked hypertrophy and enlargement of the right side of the heart, and for a time better compensation exists than in mitral disease—more dilatation is united to the hypertrophy.

An auxiliary symptom of much importance consists in an accentuation of the pulmonary artery second sound. This is obviously a symptom of the increased pulmonary blood pressure, and a comparison with the pulmonary sound of health, or with cases of mitral regurgitation, leaves no doubt of the accuracy of the statement. The first sound may also become so valvular that it can easily be confounded with the *second* sound, were we not guided by the ventricular impulse. Two explanations of this accentuation may be offered. By one, we note that the blood pressure in the right ventricle is increased relatively with the elevation of the blood pressure in the pulmonary circulation, so that the tricuspid valves close with an intensified sound, audible over both ventricles.

By the other hypothesis, if the obstruction be extreme, very little blood reaches the cavity of the left ventricle at the time of the systole. We must, therefore, subtract from the first sound the blood element, and a portion of the muscular element, leaving either a valvular sound, as heard in typhoid fever, or a dull, blurred sound, as the valves may be much more thickened than roughened.

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* *See* article on Hypertrophy and Dilatation.

Reduplication of the first and second sounds is an occasional concomitant, explicable by the fact that the overfilled right ventricle and pulmonary artery lead to right ventricular, or pulmonary artery systole, prior to left ventricular or aortic systole. It has been said that auricular and ventricular hypertrophy is more thoroughly compensatory than in cases of mitral insufficiency, and the compensation lasts longer. While this is true, transitory respiratory embarrassment, due to variations of the pulmonary circulation, are more frequent than in mitral regurgitation. The associated dyspnœa becomes more severe if the compensative power is exceeded, because the lesion is, *de facto,* more grave. The pulmonary capillaries are habitually overcharged with blood, so that slight increase in pulmonary blood pressure produces serious effects. Bronchitis of asthmatic type and cardiac asthma are frequent concomitants. The general series of phenomena outlined in the clinical history incident to mitral disease find a parallel in the history of mitral obstruction.

Palpitation and dyspnœa are, perhaps, more common than in other forms of organic heart disease. The causation is variously explained; when the facts are nucleated, the pathology of the symptomatology seems to be, fluctuations of vaso-motor tonus. The vaso-motor tonus is profoundly influenced by causes operating on the nervous system directly; for instance, the excitements of business, emotional, psychical disturbance of any kind. *Functional* digestive disorders have an important influence upon these conditions. To thoroughly comprehend this, we must recall that nitrogenous food is taken up by the liver, and therein broken up from the form of peptones into glycogen, and the products of the urea series. The excess beyond the purposes of nutrition cannot be excreted by the kidneys as rapidly as they are supplied by the liver. In the blood-vessel system these materials act as irritants, producing a recognizable increase in the blood pressure.* Dr. Parks has performed some experiments relating to this subject upon the soldiers at Netley Barracks, England. In those experiments it was found that if nitrogenous food was withdrawn from their diet, the arterial pressure was sensibly reduced; on the other hand, it became elevated just in proportion to the increase of those articles of food containing nitrogenous substances. Imagine a dyspeptic individual with mitral disease suddenly seized with palpitation and dyspnœa. Congestion of the lungs ensues, hæmoptysis, perhaps bronchitis with œdema. If in addition, renal activity is deficient, elimination of nitrogenous material is reduced, and the embarrassment of the circulation is increased. This is the explanation of the temporary dyspnœa which so often puzzles the observer, since the phase of the cardiac or pulmonary condition seems inadequate to explain the grave symptoms. Drs. Albutt and Johnson have assigned essentially the same explanation for the transient dyspnœa attending cases of nephritis, in which there is no dropsy to account for the condition. It has been called by them uræmic asthma†

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* *Lumleian* Lectures, 1877; also *British Medical Journal*, September, 1877.

† *The explanation* as above given is applicable to many cases of functional *palpitation.*

Disappearance of Presystolic Murmur.—In certain cases the murmur of presystolic rhythm may vanish, to reappear, perhaps, in a few days. Position also influences the development of mitral obstruction. They can be heard in the upright, but not in the recumbent posture; sometimes the reverse is true. Rest in bed may render a murmur temporarily inaudible. The reason of this phenomena is intertwined with a study of the blood pressure in the pulmonary circulation and the heart. *The pulse of both mitral* obstruction and regurgitation is apt to be small in volume, but in mitral obstruction the percussion wave is smaller than in mitral regurgitation, because there is less hypertrophy of the left ventricle. The pulse is also frequently very irregular in rhythm, especially during attacks of pulmonary congestion. The irregularity in these cases is probably due to a want of synchronous action between the right and left ventricles, due to the repletion of the former. (*See* Irregularities of Rhythm.)

Double Mitral Murmur.—Mitral obstruction and regurgitation, with double see-saw murmur, may be present. The lesions, however, inducing each murmur usually occur separately. Nevertheless in mitral regurgitation, when there is extensive loss of valvular structure in the mitral valve, the auricular surface may also be involved, and the murmur be double.

CHAPTER IV.

AORTIC DISEASE. OBSTRUCTION. REGURGITATION. VALUE OF THRILL IN DIAGNOSIS OF VALVULAR DISEASES.

In the outlines of the etiology of valvular cardiac disease, aortic lesions were linked with *persistent* high arterial tension, with atheroma, or with the calcareous or fatty degenerative changes of advancing life. It was not designed to affirm that aortic lesion never follows rheumatic endocarditis. So far from this, roughening of the ventricular aspect of the aortic valves and stenosis are quite frequent as the sequel of endocarditis, and since any aortic valvular disease tends to shrivel and shorten them, they may become incompetent to close the aortic orifice, and aortic regurgitation may follow. Our remarks were intended to show why the mitral valve should most frequently suffer in endocarditis upon mechanical principles. The valvular lesions, anatomically considered, are essentially the same as those developed in the mitral valve; including, on the one hand, roughening, or stenosis, on the other, such valvular destruction as shall result in rendering the opposition of the semilunar segments impossible, and regurgitation from the aorta into the ventricle results. The lesions terminating in stenosis, or roughening of the ventricular aspect of the valves, usually tend to develop insufficiency and regurgitation, with a double murmur as the result. The lesions can, however, occur independently, but the murmur of aortic regurgitation is less common as an independent murmur, than that indicating aortic stenosis. We have then two murmurs connected with the aortic region, one clearly systolic, one at the beginning of the diastolic period, called diastolic. *Systolic* aortic murmurs are carried upward by the blood current to the place at which the aorta is most superficial, and are heard with maximum intensity at the second right costal cartilage. From thence the murmur is propagated into the carotids, and can in exceptional cases be heard even in the femoral, radial, or dorsalis pedis arteries. Aortic systolic murmurs are timed by the same methods we employed to time systolic mitral murmurs. To distinguish them from a systolic mitral, we have the different location and line of transmission, with maximum intensity. In the case of single aortic, or mitral systolic murmurs, the diagnosis is easy; for instance, the latter are not transmitted into the arteries of the neck, and the former diminish in intensity as one listens toward the axilla. But aortic murmurs are very widely transmitted, the sternum acting as a sounding board, and aortic murmurs can be distinctly heard over the sternum, at the epigastrium, in the axillæ, or at any part of the chest when the murmur is rough, or loud. When both mitral and aortic systolic murmur co-exist in the same case, it is very important to locate exactly the point of maximum intensity of each murmur.

In addition, there is another fact, viz., two systolic murmurs are seldom of the same pitch and quality. Most frequently the aortic is the highest and roughest, since calcareous degeneration, and atheroma, of bony hardness, is more frequent at this point.

Diastolic Murmur.—The diastolic murmur indicative of aortic regurgitation can be heard at the base, but is manifestly louder as the auscultator nears the aortic cartilage, which is the place at which that vessel is nearest the ear. Aortic regurgitant murmurs are transmitted in two directions. This happens because the blood current is flowing onward through the arterial circulation, and also by reason of the lesion backward into the ventricle. Moreover, in the latter case, the sternum conducts sound very readily, so that the murmur can be heard loudly at a point just above the ensiform cartilage. In fact the mid-sternal line at the articulations of the sixth ribs frequently presents a second centre of equal intensity. The murmur can sometimes be detected in the carotids, and along the aorta, but always with lessened intensity as the auscultator leaves the heart. Behind the sternum lies the bulk of the heart, and mostly the right ventricle, but at the apex the left ventricle is superficial, and aortic regurgitant murmurs can sometimes be heard at the apex.* Like aortic systolic murmurs, diastolic aortic murmurs are distributed over a wide area, and in many cases can be heard in the axillæ of the right and left side. The pulse of aortic regurgitation is very diagnostic. It has been called the trip-hammer pulse, or the Corrigan pulse. When the aorta is normally distended by the aortic systole, there is a gradual aortic recoil, or systole which aids in forwarding the circulation. But if the valves are incompetent, the aortic systole forces the blood quickly backward into the ventricle, as well as forward through the aorta, and the arteries become abruptly, abnormally empty. Since the ventricle is usually hypertrophied, the percussion stroke of the pulse resembles that of a ball striking the finger, and quickly rebounding. It is represented by the sphygmograph in the accompanying figure.

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* *See* record of two cases in which aortic regurgitant murmur was heard only at the apex. One was a case of rheumatic endocarditis; the other a case of valve rupture. Dr. Balthazar Foster suggests that a murmur heard as above indicates rupture of the posterior aortic segment. The other two segments being situated below the mouths of the coronary arteries; when they are diseased, the diastolic blood column is either prevented from entering them freely, or runs past their mouths into the ventricle again, and cardiac nutrition must be more directly affected, than if the other segment of the semilunar valve is diseased.—*Lancet*, Aug. 14th, 1869, p. 225.

Its characteristics are the abrupt percussion stroke, the rapid fall with exaggerated dicrotic curve. In the Philadelphia *Medical Times*, May 21st, 1881, three cases of Corrigan pulse are reported in aortic dilatation without valvular lesion; but the pulse may be said to be almost diagnostic. The special character of the Corrigan pulse is much increased by elevating the arm above the head.

The pulse of the aortic obstruction is sometimes of small volume (although the cardiac action is strong) if the obstruction is extreme, but oftener by a pulse full, strong, and hard, as an evidence of the ventricular hypertrophy. The local *consequences* of aortic disease are observed chiefly in the condition of the left ventricles. This cavity bears witness to a constant effort on the part of nature to secure compensation. For the most part these efforts are successful, and in both forms of disease hypertrophy of the left ventricle is a *marked* conservative lesion. The strongest possible contrast is presented to the cases of mitral disease in which hypertrophy cannot be so efficiently conservative. The hypertrophy continues for years without being followed by dilatation, even in the face of considerable physical exertion. This is also in strong contrast with the effect of labor in mitral disease. Old age is often attained by those suffering from aortic disease without much discomfort; but the advent of preponderating degenerative changes is insidious and sure. To fully appreciate this, consider that in aortic obstruction, the aorta being imperfectly filled, the coronary arteries supplied by the aortic systole (in its turn dependent on complete arterial distension), are imperfectly supplied, and fatty degeneration slowly commences. The same changes proceed in aortic regurgitation; in this case if the portions of the valves directly beneath the coronary arteries are more perfect than other portions of the valves, the percussion of degenerative changes is slower, since the blood is more or less efficiently directed into the coronary arteries. If the reverse prevails, or the mouths of the coronary arteries become more or less occluded, the course of dilatation, fatty degeneration, is more rapid. Another insidious source of danger is atheroma of the coronary arteries; this process begins as the result of increase or aortic systole, induced by the violence with which the blood is forced from the ventricle into the aorta.

With mitral disease death stalks behind the complications; in aortic disease death results immediately from the condition of the cardiac muscle.

*Associated Lesions, and Symptomatology connected with Aortic Disease.**—Lesions of the aorta itself occur both as the harbingers or as the result of valvular aortic disease. In either case increased arterial tension is the initial factor; on the one hand, due to cardiac hypertrophy, on the other, to causes which will be developed in the section on aneurism. In a small proportion of cases atheroma of the aorta is simply a degenerative process incident to advancing life. Indeed, symptoms of aortic lesion are intertwined with the symptomatology of aneurism, combined with hypertrophy of the heart, and cannot often be separately studied. The leading symptoms are: pain, due to pressure of the enlarged heart or aorta on adjacent tissues or nerves, palpitations, and various anginas, doubtless symptomatic of changes in vaso-motor tonus. Apoplexy, either embolic or due to coincident atheroma of the arterioles of the brain, is a frequent complication. The rapidity of the fatty degenerative changes are directed by the same general conditions noticed in the chapter on the fatty heart.

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* *See* Hypertrophy and Dilatation.

The dropsies and symptoms of venous congestion are not common in aortic disease until the terminal stages.

Mitral Lesions a Sequence of Aortic Disease.—The reflux of blood against the walls of the ventricle, in its effort to find exit through a diminished aortic orifice, increases the pressure on all parts of the ventricular wall, which will naturally yield at its weakest point. The pressure is increased by the violence of the contraction, owing to the concomitant hypertrophy. The yielding may occur at the mitral orifice, permitting regurgitation, and is really a compensatory and beneficial lesion, since it relieves the over distended ventricle.

General Considerations.—There are a few facts connected with valvular lesions not yet alluded to in detail: One is palpitation, one is thrill, another violent carotid pulsation, finally, the relation of murmurs to sounds. Carotid pulsation is an incident of valvular aortic disease, or violent carotid pulsation may indicate hypertrophy or over action of the heart without valvular disease. It is common, also, in diseases affecting coats of the vessels, especially the aorta. *The relation of murmurs to sounds* is such, that murmurs may replace the sounds, or a more or less normal sound may persist. In the latter case some valvular competency is probable, but most frequently the murmurs destroy the normal sound. The partial abrogation of a cardiac sound occurs mostly at the aorta. In aortic obstruction especially, the second sound, though perhaps modified, persists, unless there is regurgitation. *Thrill* is a purring tremor or vibration communicated to the hand in the præcordial region. It is an analogous tremor to the pulmonary vibration, "the vocal fremitus." Thrill can originate in the pericardial sac, and is then known as a pericardial friction.*

Thrills are also developed *within* the heart or the great blood vessels. Endocardial thrills are coincident with the systolic or diastolic periods. Endocardial thrill is developed by the whirling of the blood stream over roughnesses on degenerated valves, or at contracted valvular orifices, or again by the roughnesses in the walls of the great vessels. The vibration which is felt as a thrill is also audible as a murmur, but only a small proportion of these disturbances are sufficiently violent to be sensible to palpation, so that very few audible murmurs are associated with thrill.

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* *See* Pericardial Friction Sounds.

They follow the same laws of location of maximum intensity as the audible murmurs. Practically speaking mitral obstruction is the lesion in which most commonly the interference with the normal circulation is manifested *both* by murmur and thrill. The location of maximum intensity in such cases is the apex. The time, post-diastolic or presystolic. Thrill, connected with mitral regurgitation, is far from common. At the base of the sternum, thrills may proceed from the aorta, or the right ventricle.

Systolic and diastolic thrills can be recognized over the aortic region, their presence (excluding pericardial friction) indicates atheroma, aortic dilatation or saccular aneurism. The violence of a thrill, as of a murmur, can be increased by exertion. By combining the inclusive evidences of disease, the adjudication of the import of thrill is easy; for from what has been said, it must be deduced that thrill is by no means specially diagnostic, but is oftenest associated with mitral obstruction or aortic disease.

Thrills detected in the veins of the neck indicate a narrowed calibre from pressure. In regard to palpitation, we refer to the section on functional heart disease, and to the discussion of palpitation connected with mitral obstruction, since it is not a symptom frequently incident to the majority of valvular lesions.

CHAPTER V.

ANEURISMAL DISEASES OF THE ARTERIAL SYSTEM.

Lesions of the aorta of moderate extent are so frequently associated with diseases of the valves as just described, that it seems in place to follow the account of the lesions of the valves with an account of aortic disease.

It is convenient to nucleate the lesions of the aorta into the following classes:—

1st. Simple dilatation of all the coats of the vessel.

2d. Saccular Aneurism.—The forms of the sac very various.

3d. Dissecting Aneurism.

The first two conditions will alone claim our notice in an outline of diagnosis of thoracic affections. Aneurismal changes are always associated with, or preceded by a diseased condition of the contiguous layers of the internal and middle coats of the vessel, a tissue growth terminating in degeneration, which by impairing the elasticity, and contractility of the walls of the vessel, allows of their expansion and dilatation, even under the tension of the normal arterial blood pressure, or this abnormally increased by any cause, such as occupation, which acts as a fostering agent to the seeds already sown by the structural changes. The structural change is in association, in a major number of instances, with syphilis, and in the order named with the other causes of atheroma ("Endo-arteritis," "fatty degeneration,") viz., intemperance, Bright's disease, rheumatism, advancing life.

Of the physical signs of atheroma without much dilatation, there are none concerning which we can feel sure. Calcification of the arch is attended with a rough systolic murmur, with a location of *maximum* intensity limited to the vessel.* In contrasting systolic aortic valvular and systolic aortic murmurs, questions, we think, are adjudicated by the axiom that murmurs are heard with maximum intensity at the portion of the circulatory tract where the cavities in which the murmurs are generated approach closest the chest surface. Thus a systolic murmur becomes indicative of associated aortic valvular and aortic lesion when heard with equal intensity over the aorta as over the valves, *i. e.* the second right costal cartilage, if heard with maximum intensity over the aorta only, is strikingly indicative of aortic lesion. Since the procession of causes of valvular disease are similar, we often do have associated valvular disease. So far as hypertrophy or dilatation are concerned, both in a marked degree accompany valvular lesion. In atheroma the aorta is unyielding, and the effect of its resiliency on the circulation is lost, and the heart becomes somewhat hypertrophied to meet the demand; but in aneurism, or atheroma, the heart is never so much enlarged as in valvular aortic disease. The general symptoms possess grave import, since they tell of the ultimate effects of a rigid arterial system; in the anginas, the obliteration of small arterial branches by calcareous degeneration, or by the formation of thrombi.*

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* Murmurs indicative of atheroma, or aneurism, are sometimes telephoned into the smallest branches of the arterial system. They can be heard sometimes in the radials, or the dorsalis pedis arteries.

We use the word *aneurism in its widest sense as a local increase in the calibre of an artery* Thus any form of aortic enlargement can give rise to some or all of the physical evidences of aneurism, which are purely mechanical. The physical evidences depend upon two conditions. *First,* the size and direction of the growth determines the pressure symptoms. *Second,* the nature of the sac, and the variety of orifice by which it communicates with the parent artery, are conditions which give direction to the acoustic phenomena.

The evidences of aortic dilatation are headed by percussion. Practically, whenever the aorta is dilated, there is dullness over the upper piece of the sternum, between the third rib and the notch; there is also a broadening of the area of dullness laterally, encroaching materially upon the lung, usually to the right, but often also to the left of the sternal border. An excellent method by which to demonstrate this, consists in causing the patient to make full inspiration, noting that the percussion, previously dull, has become resonant; then cause full expiration to be accomplished, and note that the dullness is rendered more pronounced than it was in quiet respiration. The result is evidently due to the fact that the edges of the pulmonary lobe circle the aorta on full inspiration, and are withdrawn and the aorta exposed by expiration. The method of percussion is the same advised for examining the heart: commence in an interspace quite outside the area of impaired resonance; gradually approach the sternum until the area of impaired resonance is fixed.*

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* Apoplexy, dyspepsia, fatty degeneration of organs, notably the heart, atrophic emphysema, are some of the conditions accompanying arterial changes.

Palpation.—Thrill or fremitus may or may not be present; the sign is dependent on the physical condition of the wall of the artery. It should be sought for along the chest wall above the track of the vessel, and also in the sternal notch; and since the innominate and carotids may be also atheromatous, the thrill may be transmitted in them. Pulsation is another evidence sometimes but not always present. The pulsation can be made more evident by causing the patient to incline forward, so as to bring the aorta as closely as possible in relation with the chest wall. Search, then, by palpation, not only over the upper piece of the sternum, but also at the sternal notch. There will be a jerking, inelastic impulse behind this point. The aorta cannot ordinarily be felt over the sternal notch, but if it should be perceptible, the pulsation, to be indicative of atheroma, should posess the above named peculiarities. Palpation will include the study of the pulse. There will be often associated atheroma of the radials more or less marked;† the qualities of hardness, suddenness, jerkiness, are very striking. In some measure the Corrigan, or pulse of aortic reflex, is simulated; "even the hooked apex of the sphygmographic tracing of aortic insufficiency is found in the rigid pulse of atheroma and calcification" (*Walsh*).‡

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* Naturally, the area of dullness depends upon the direction of the enlargement. The tumor may occasion dullness, which can be detected posteriorly as well as anteriorly.

† The palpation of atheromatous radials yields a sensation similar to that of a piece of ipecac root under the skin.

‡ *See* also *Med. Times*, for May 21st, 1881, cases presenting Corrigan's pulse without aortic valvular lesion.

Sometimes the orifice of the innominate or pulmonary artery is more or less occluded by spiculæ of calcareous matter, or the lengthening of the tube due to the disease may reduce to a slit-like form the orifices of the arteries named, or direct globular enlargement may take place, more or less altering one or other orifice. The outcome of the study amounts to this, that the pulse may vary materially in volume on one or the other side, from the above causes, but unless there are associated local evidences of disease, we are not justified in locating an aneurism on one or the other side by the qualities of the pulse. Inspection merely confirms the results of the previous examination; local bulging of the tissues, visible impulse of the carotids of supra-sternal pulsation, can severally be observed.

The varieties of saccular aneurism are capable of modifying the physical signs as follows:—

Inspection and palpation may disclose the shape of the tumor itself. It may grow outward and project as a mass, even as large as a small cocoanut. The walls may be gradually reduced to the thickness of the integument, and the merest shell of the external coat. The pulsation and thrill are detected, in the writer's experience, with a readiness proportionate to the thickness or thinness of the walls of the sac.

Auscultation.—The question of the causation of murmurs is fully discussed in books devoted to the subject of diseases of the heart only. Let it suffice for these outlines to say that murmurs may be systolic or diastolic, or both, in the same case. It is probable that the entrance of blood into an aneurism may be simultaneous with the systole, the exit of the blood synchronous with the diastole. Usually the murmurs indicative of aneurism of the true or sacculated variety have the hollow ring called bruit, the analogue, in this instance, of the cavernous tone imparted to respiration when the air enters a cavity in the lungs. The bruit is a hollow, reverberating murmur, usually longer than the other murmurs thus far described. Once heard, it can afterwards easily be appreciated. A single practical demonstration is worth pages of adjectives. Whether the murmur be single or double depends almost entirely upon the nature of the orifice. Sometimes, in cases of aneurism of the cœliac axis, the orifice may be merely button-hole shaped, or the orifice may be abrupt or smooth. A murmur, when very prolonged and systolic, indicates simple fusiform dilatation, or that the tumor communicates with the aorta by a very small opening (*e. g.*, aneurisms of cœliac axis); or else that the tumor is largely filled with clots of fibrine. The latter point is confirmed, if the murmur is more muffled than usually noted in aneurisms containing blood, which are relatively equally, superficially or deeply situated.

More important than these questions is the fact that aneurisms may exist *without the suspicion of a murmur;* or there may be no murmurs, properly speaking, but only a dull, impulsive impression, systolic in time. Sometimes the second sound is natural, sometimes it is accentuated.

In early cases of aneurism, before marked bruit exists, aneurism must be diagnosticated by other means, or by a method of study to be suggested presently.

If the aneurism be filled with clot, especially if the clot be laminated, very little blood can enter or find exit, and thus very feeble murmur is developed, since the orifice of a sac communicating with the aorta may be reduced to a mere depression. In a case of atheromatous fusiform dilatation we have seen a large clot so fill the dilatation that no murmur was generated during life. The depth of the plane at which the murmur is generated may be of service in forming an opinion as to the nature of the contents of a sac. The more distinct and superficial the murmur, the more likely is the sac to be but partially filled with clot.

In some cases, if the tumor be superficial, the pressure of a stethoscope may influence the lumen of the aorta, and so develop a murmur not otherwise present.*

Abdominal Aorta.—In examining the abdominal aorta, there are some additional points to be considered. It is much more convenient to invoke palpation to our aid. Aneurisms in general are accompanied by an expansile pulsation, which is very diagnostic of aneurism when it can be felt. This is applicable, in an especial degree, to abdominal aneurism, and also in thoracic aneurisms, pointing externally.

Care must be observed not to confuse a transmitted pulsation imparted to a tumor in the abdominal cavity. The hand and knee position will obviate, in many cases, a transmitted pulsation, but an aortic enlargement will continue to pulsate distinctly.

In abdominal aneurism the position upon the face or side gives more ease than the recumbent posture on the back. This is because the throbbing pressure of the tumor upon the vertebræ is mitigated, and this pressure is a source of great pain.

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*As representative of many cases of aneurism without murmurs, see *Medical Times and Gazette*, Jan. 31st, 1880 (Broadbent).

The posture becomes a great aid to diagnosis in cases of pain in the back in which aneurism is suspected. In some cases lancinating pains run round the abdomen to the anterior parietes, giving the sensation of a cord around the body.

A throbbing pulsation of the lower part of the abdominal aorta is a symptom not infrequently met with, especially in hysterical women. It must not be mistaken for a diseased condition of the vessel.

Auxiliary Methods for the Study of Aneurism.—We wish now to indicate some of the other collateral symptoms of aneurism and a method of study. In methods of investigation, the question of etiology is fundamental as a predisposing condition.

1. Laborious avocations act the part of exciting causes second to the predisposing conditions already cited. 2. The pressure symptoms occupy a leading place in view of the already determined etiology. The etiology and the location of the pressure symptoms will usually suffice to differentiate aneurismal pressure symptoms from other tumors. Saccular aneurism is a disease of early life; it chiefly occurs between the ages of twenty-six and forty years, after which the lesion will oftener be of the variety known as equable dilatation of the aorta. Most of the causes of aneurism find expression in the early period of life; in latter years atheroma is a sequel of the fatty degeneration of advancing years. Pressure symptoms are determined by the location of the growth; in thoracic aneurism, if the tumor enlarges inward, the pressure symptoms are noticed by functional incapacity of œsophagus or trachea. The vertebræ undergo atrophy, or necrosis; the intercostal, the pneumogastric, the sym-

pathetic ganglia in the neck, are all involved. In the latter case the irritation results in stimulation sufficient to occasion dilatation of the pupil of the affected side. The laryngeal nerves are involved, especially the right, which curves around the aorta, and laryngeal irritation or paralysis ensues. A strongly important point, even in cases where there is but partial paralysis, is the *harsh laryngeal bark*—it is very pathognomonic of laryngeal irritation. It is heard in tumors of the larynx, but should always lead to examination of the aorta. Moreover, if the larynx is lifted upon the forefinger inserted in the cricoid under the thyroid cartilage, we can often feel pulsation communicated to the finger by the enlarged vessel. There is one pressure symptom cardinal in its importance; this symptom is pain. It is persistent, and yet it can be mitigated by changes of attitude, which indicates that the pain has a mechanical origin. To illustrate this point, we have in the case of abdominal aneurism very marked mitigation of the pain when the prone posture on the face is assumed.* Pain is caused partly by necrosis or atrophy, the result of pressure upon the sternum or vertebral column; partly by pressure or irritation of adjacent nerve trunks. The pressure upon the nerves gives rise to the variety of pain known as neuralgia.

The proposition then is, that by the correlation of the etiology, the pressure symptomatology, and the physical examination, the diagnosis of aneurism can in most cases be made as unequivocally as in the more simple instances of disease.

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* In thoracic aneurisms, for instance, if the sac impinges upon the trachea the patient steadily keeps the head forward, or perhaps sideways, or throws back the head while partly sitting in bed, in the effort to relieve pressure upon the windpipe.

Even in a brief manual it is desirable to study the *natural history* of aneurism, in view of its terminations.

Solution of continuity of the walls of the sac may be effected by slow perforation or sudden rupture. A sudden rent through the thinnest portion of the sac may be instantly fatal, by hemorrhage. Or, the stratified layers of the aneurism may be perforated by a very minute stream of blood, and several hemorrhages may occur, preceding the final issue by a very variable space of time.

There is hardly a conceivable situation at which hemorrhage has not occurred. If internal rupture happens, the pericardium, the pleura, the mediastinum, the stomach, the abdominal cavity, all may receive the contents. The blood may be voided by the mouth if hemorrhage occurs into the trachea or bronchus, the lung substance or the œsophagus. The interesting fact in this connection is, that hæmoptysis from aneurism has been mistaken for the hæmoptysis of phthisis; of course, in such cases the rupture of the sac was by slow perforation into a bronchus. Death also occurs by exhaustion from pain, insomnia, or the dyspnœa and asthma due to broncho-tracheal pressure.

It is an interesting fact, and one of the greatest value in the differential diagnosis from malignant tumors of the mediastinum or abdominal cavity, that wasting of the tissues is not a marked accompaniment of aneurism, unless the anguish suffered induces anorexia, with deficient power of assimilation and elimination, and possibly fever.

Aneurisms of the pulmonary artery are very rare, probably because the antecedent of aneurism (atheroma) is not likely to occur in this vessel. The usual signs of aneurism, pulsating tumors, thrill, murmur, are present,* but in addition there is lividity of the face, dyspnœa and a pulse of small volume. The differential diagnosis must be considered between aneurism of the aorta, or a hypertrophied or dilated left auricle.

Pulsations of the Arteries without Aneurism.—In the second left intercostal space, close to the sternum, one can sometimes find a pulsation which is diastolic in rhythm. The pulsation arises in the pulmonary artery, and is due to the repletion and distention of that vessel, and consequent exaggerated recoil. It is sometimes a feature of hypertrophy of the right ventricle, in its turn a sequel to mitral obstruction or regurgitation. A diastolic impulse in the right second intercostal space, close to the sternum, is a symptom of some aortic dilatation. But as the lesions giving rise to aortic disease are liable at the same time to induce valvular disease, regurgitation prevents the pulsation of the aorta from being a constant incident of aortic dilatation.

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* *See* Introduction, for position of pulmonary artery.

CHAPTER VI.

INORGANIC OR FUNCTIONAL MURMURS IN THE HEART; THE VENOUS, THE ARTERIAL SYSTEMS.

We have still a class of murmurs not grouped among those already described. They include functional or inorganic murmurs, venous murmurs, and arterial murmurs, developed with and without atheroma.

Inorganic murmurs are sometimes developed over the heart in the course of the zymotic fevers, in chronic Bright's diseases, in leucocythæmia—in a word, in any of those conditions in which the crasis of the blood is seriously reduced. There is ground for the belief that these murmurs are associated with the quality of the blood tissue, since they disappear as soon as the anæmia is overcome. Inorganic murmurs are also sometimes developed by coagulation of blood within the heart. Their recognition involves the following considerations: Organic murmurs speedily induce alterations in the walls of the various cavities of the heart, which find their expression in abnormal centres of pulsation, changes in percussion resonance, etc. Organic murmurs cause an accentuation or weakening of the other cardiac sounds, according as they increase or diminish vascular tension in the various cavities. In the case of inorganic murmurs the cardiac sounds are normal or feeble, only accentuated if fatty degeneration is present. Moreover, instead of having a fixed centre of maximum intensity, the centre of loudness varies; now these murmurs can be heard at apex, now at ensiform cartilage, now at the aorta. Since they are independent of local lesion, they follow the axiom that all murmurs find their centre of maximum intensity at the locations in which the cavities in which they are generated approach closest the chest's surface. The murmurs are sometimes produced in the right side, sometimes in the left side, of the heart; hence the above variation. They are commonly heard most distinctly at the base, the place at which the valves are situated in near relation with one another, and also the point nearest the origin of both pulmonary artery and aorta. Inorganic murmurs are in direct association with the etiological states to which they owe their existence.

Again, inorganic murmurs, while they may be transmitted into the arteries, are often reinforced in intensity as one recedes from the heart. If one causes the patient to turn the head to the side opposite the one examined, a humming-top sound, or venous murmur, the *bruit de diable* may be heard. This makes it very probable that the arterial sound is hæmic. Finally, the quality of the murmurs is soft and blowing. Sometimes they occur as a humming noise, and they are always systolic. In regard to the possibility of thrombi developing a murmur, the same points will apply, save that their genesis does not require the association of the anæmic state. In thrombosis, their sudden advent and the shifting location of maximum intensity are two valuable points. In addition etiology will be helpful. (*See* Thrombosis.)

Venous Murmurs.—When the stethoscope is applied to the neck a distinct humming sound is often to be heard. This venous hum is usually continuous. It can be heard most unequivocally in persons who are profoundly anæmic; also in cases of inflammation of the venous coats, or pressure on a vein. The veins of the neck are almost solely the seat of murmur, and it is, perhaps, desirable to record some instances of pressure. For example, pleuro-pericardial adhesions, or tumors of the mediastinum, can occasion pressure on the veins. The position has much to do with the production of murmur, and it has been suggested that murmurs should be best heard in the erect posture, since the blood current is then at its swiftest. It may also be louder during inspiration, since this act markedly accelerates the flow of the blood toward the chest. These murmurs can be developed in a perfectly healthy person by pressure of stethoscope, a point of extreme practical importance.

Arterial Murmurs.—These are developed solely by such pressure as can diminish the calibre of an artery, and so impede the blood current. The most important is the so-called subclavian murmur, which can be heard in cases of *incipient phthisis,* in the subclavian arteries. It is usually a blowing, somewhat high-pitched murmur, yet one which is rather soft than harsh in quality. It is due to the pressure exercised by early consolidation upon the calibre of the artery, or it may be due to the pressure of pleural adhesions, which sometimes accompany early phthisis.

Arterial murmurs due to atheroma can sometimes be heard in the superficial arteries, especially in the subclavian or axillary arteries; in these cases, an examination of the radial will reveal atheroma. Atheroma can be *detected* by the hard, unyielding character of the vessel. It resembles a piece of ipecac root beneath the skin. It is possible that in states of reduced arterial tonus arterial bruit can occur from the simple vibration of the walls of the vessel after the cardiac impulse, causing murmur; but this is scarcely a practical question, as arterial murmurs are accompanied by anæmia, unless due to the causes previously mentioned.

It is impossible, in the limited space at the writer's command, fairly to discuss questions of murmur, functional or organic, connected with the right heart, and arising from congenital lesion. The reader is referred to systematic works on this subject, since the question is of theoretical rather than practical interest.

Endocarditis without murmur possesses a weird outline, hardly consistent with positive diagnosis. It includes many of the symptoms mentioned in the pages devoted to changes in cardiac rhythm. The sounds become irregular, or reduplicated. There is more or less shortness of breath, pain, headache—in fact, since the disease is most frequently associated with acute articular rheumatism, with Bright's diseases, or pyæmia, the blood lesion finds expression in a most varied guise. When murmur is present with an environment favoring endocarditis, how are we to decide whether the murmur is old or recent? Murmurs of long standing produce changes in the heart substance and cavities. There is a history of prior dyspnœa, or palpitation. The formation of clots and embolism are exceptional features of acute endocarditis. For the diagnosis of cardiac thrombosis, see appropriate section.

Ulcerative endocarditis is a rare disease, sometimes occurring in pyæmia or blood-poisoning. Ulcerations on the valves, or extension into the muscular substance of the heart, may cause cardiac aneurism or rupture.

Embolic pneumonia, metastatic abscess. The disease is marked by the associated symptomatology of pyæmia, most prominently by remittent or intermittent febrile thermometrical *record, with rigors*, followed by sweats.

CHAPTER VII.

FUNCTIONAL DISEASE. VARIATIONS OF RHYTHM. NEUROSAL DISEASE.

In treating of the diagnosis of neurosal functional disorders of the heart's action, we are led to say that a diagnosis by exclusion of the diseases of the heart already enumerated, should be a primal step. The symptomatology is so various, we cannot hope to reduce an adequate description within the scope of these pages. The etiology is a subject, in itself; so manifold, and protean are the influences, one is reminded of the fable of the sowing of the dragon's teeth. Insignificant causes broaden out into serious functional inefficiencies. We naturally take cognizance of such habits as excessive indulgence in tobacco, in alcohol, in sexual appetite. Behind all these are psychical influences,which profoundly influence both vaso-motor tonus and the innervation of the heart directly. The investigator must lift the veil of each separate case by individual penetration, and practice of both the science and the art of medicine.

We observe in the outset, that many of the symptoms of "heart starvation," or fatty heart, may be intertwined with functional disorders without demonstrable cardiac lesion. In the group of functional disorders we shall allude merely to deviations of rhythm, and the symptoms of dyspnœa and pain. Deviations of rhythm include *intermission* and *irregularity*.

Irregularity of rhythm and intermittency are conditions which differ only in degree. In intermittency the pause between the pulses is longer, there being an interval equal to that occupied by a pulsation. Irregularity is a want of accord in the action of the layers of muscular fibre of which the heart consists. The head does not wait for a whole beat, as in intermission, but alters its rhythm irregularly. If the right heart does not complete its contraction at the same instant as the left heart, then the action may be reduplicated. Sometimes the periods of action and rest cannot be discriminated, and only a tumultuous throbbing is recognized. Patients sometimes describe this as a fluttering action of the heart. These conditions exist, as we have said, independently of organic disease. For instance, psychical causes, strong emotion by terror, anxiety, grief, pain, fatigue, indigestion, use of tobacco, sexual excess, may cause irregularity, or it may be congenital. On the other hand, these variations of rhythm are very representative of fatty degeneration, or dilatation. The adjudication of the import of these signs is chiefly based on the position and strength of the impulse as felt over the præcordia. If notable feebleness of impulse co-exists with irregularity, and especially insufficient systole, the import is serious, since it may indicate fatty degeneration and dilatation. It may then be a sign of degeneration of the left ventricle, in which case the ordinary stimulation of the blood with which the auricular systole supplies the ventricle is inadequate, and a pause occurs until a second or third contraction supplies it with more. It is of especially serious import, if associated mitral disease or regur*gitation exist.* The most crucial test depends upon the effect of *effort* on the heart. An irregularity deduced from causes which are neurosal is scarcely affected by effort—the pulse may quicken, but its irregularity is often rather diminished than quickened. When the irregularity is due to valvular disease, or degeneration, a very slight effort, a brisk walk up and down the room, noticeably increases the irregularity.

Reduplications.—The heart sounds occur more or less doubled or repeated; sometimes one of them is actually repeated, so that we hear two sounds instead of one. It is a feature of intermittence, the sounds being reduplicated with some beats, and not with others. The normal sounds depend on the synchronous closure of the tricuspid and mitral, or the pulmonary and aortic valves. The synchronism depends very much on the blood-pressure in the cavities of the heart, or in the calibre of the pulmonary artery or aorta. If the sound is a veritable reduplication, the first sound is reduplicated at the end of expiration or beginning of inspiration.* The second, at the end of inspiration and the beginning of expiration. The condition must not be confounded with mere ineffectual systole, in which several entire revolutions of the heart occur, until sufficient arterial tension has been gained to produce a pulse; in this case the *pulse at the radials and the heart beats* do not correspond. In reduplication, usually, only one or other of the sounds are reproduced.

Dr. Hayden says (1) "the phenomenon may be associated with simple neurosal functional derangement of the heart, or accompanying anæmia; (2) with attenuation and weakness of the ventricles in persons of middle age, of nervous temperament; (3) with a weak degenerating heart, and dilated atheromatous arteries; (4) with simple hypertrophy of the ventricle."

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* For review of theories of causes of reduplication, *see* Dr Sansom on "Physical Diagnosis of the Heart;" also *Medical Times* and Gazette, 1877, on "Reduplication of Heart Sounds," by Dr. Barr.

"Guttman says it may occur temporarily in perfectly healthy persons; it may be noted at times in diseases of the heart, sometimes connected with mitral, sometimes with tricuspid disorder, but cannot be said to be characteristic of any particular affection." It may be a symptom in the course of typhoid fever, and it may be a symptom in cases of mitral obstruction. Further, adherent pericardium leads to reduplication. The second sound is much more frequently reduplicated than the first; reduplication of the first sound is rare.

Palpitation is a common disorder of rhythm. It is evidence of a spurt of the heart to accomplish its work. Naturally it is a symptom which may indicate both organic or functional disease.

The essential pathology is probably a variation in the intra-cardiac blood-pressure; this is brought about by so many causes that it seems impossible to collate them in these pages. In the section devoted to mitral obstruction this subject was considered in some detail, and in each case of organic heart disease palpitation may be looked upon as a symptom of cardiac repletion and embarrassment. It is a frequent incident in the history of simple hypertrophy, and attracts the patient's attention, because the enlarged heart gives origin to a forcible impulse. It is, however, a very frequent symptom of functional neurosal cardiac derangement, developed by the causes originating other forms of functional disorder. We must exclude organic disease by a method of study already *sufficiently* described, and constitute each case a problem to be studied, subject to the methods indicated for the analysis of functional cardiac disorder.

Dyspnœa is a frequent symptom of cardiac disease, both functional and organic. It is also a prominent symptom of various pulmonary processes. An excellent test applicable in differential diagnosis, is the effect of exercise upon the symptom.

The patient may breathe readily while quiet, but if requested to walk briskly or ascend the stairs, if cardiac disease exist, a more pronounced breathlessness comes on. Of course an examination of the lungs should supplement the above evidence. In emphysema the dyspnœa is partly cardiac, partly pulmonary—an important point to be recognized from a therapeutic standpoint, since it indicates the use of both respiratory and cardiac stimulants for the relief of a patient. The position assumed by a patient for the relief of dyspnœa is significant. In cardiac disease the patient cannot usually recline, but must sit up during the paroxysms of dyspnœa. (*See* Cardiac Asthma.)

Occasionally, in functional heart disease, but often in organic; deficient oxygenation of the blood has the effect of producing a state of drowsiness, or even mild delirium may supervene if elimination is deficient.

If dyspnœa is an evidence of seriously deficient cardiac action, stupor may be added to the symptoms of these cases, which are usually associated with dropsy and albuminuria. The question of differential diagnosis between this phase of dropsy with uræmic symptoms, and renal dropsy with uræmic symptoms, is often raised. The cyanosis, especially of the face, and the history that the dropsy began in the more dependent portions of the body in the former case, while in the latter the face is white, the skin pallid, and the dropsy is general. We mention this because in cardiac disease the above symptoms can be efficiently met by cardiac and respiratory stimulants, combined with the use of the alkaline diuretics, or the preparations of benzoic acid, and in alkaline combinations.

Angina.—Pain in the heart is an unessential symptom of either acute or chronic heart disease. However, while this is true, pain may be complained of in any acute inflammation, such as endo- or pericarditis, or uneasiness may attend any form of chronic, functional or organic cardiac disturbance. Severe pain in the heart is sometimes felt by feeble or anæmic persons after unaccustomed exertion (*i. e.*, increased arterial tension). It may be a symptom of gastric indigestion, but may be referred to the heart, or pain may be brought on by excessive use of tobacco, or in the gouty state. Cardiac pain is differentiated from intercostal neuralgia by the painful spots in the course of the nerve in neuralgia, while pressure does not increase true cardiac pain.*

Angina Pectoris is a cardiac grip of phenomenal severity. The præcordial pain is most severe. The sensation has been described as though an invisible hand were grasping the heart, or it were being torn in pieces, and a feeling of impending death is imminent. The pain radiates down the left arm, sometimes the right, shooting to the back or neck.†

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* Pain with tenderness has been maintained elsewhere as a diagnostic symptom of intercostal neuralgia as separated from pleurisy.

† "The pain of angina is distinctly located in or about the mid-sternum, whence *it radiates. Eulenberg* says this is due to the connection between the superior cardiac nerve and the anterior branches of the four upper cervical nerves, while the

The heart palpitates somewhat, but its action is often slow and labored. The beat of the artery at the wrist may be small, irregular or accelerated, or it may be full, strong and regular, not increased in frequency, depending on condition of the cardiac and pulmonary substance. Again, there may be decided differences between the pulses, the left being almost or quite imperceptible.* The surface of the body is usually cold, and a clammy sweat beads the brow.

Summary of Diagnostic Symptoms.—1st. The attacks are paroxysmal, with long or short intervals, from a few minutes to an hour. 2d. Sense of coldness and cold sweat. 3d. Difficult breathing is not a prominent symptom. Cheyne-Stokes breathing may be present, but the breathing is always slow. 4th. The heart's action is not increased. 5th. The attack may pass off, or death may occur. The spells may be brought on by cold, fatigue, mental excitement, and are most dangerous; since the heart may pause in any paroxysm. Naturally there must be many gradations of intensity of attack, from moderate distress to anguish. The true anginæ are rare, intercostal neuralgias, rheumatism, reflex pain from dyspepsia, are common. The teaching of to-day seems to accord with the following statement. Angina pectoris is a disease associated with vaso-motor spasm, as proved by Dr. Lauder Brunton. "It is divisible into two classes; the *first* a class in which the middle and inferior cardiac nerves are connected with the four lower cervical nerves, uniting in the brachial plexus and first dorsal nerve. The pain usually runs out at the peripheral endings of the ulnar nerve, especially the little finger. It is almost invariably found on the left side only. In a case where it was found on the right side the pulmonary artery was the seat of disease."—*Diseases of the Heart, Dr. Fothergill, London,* 1879, *p.* 285.

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* Hamilton Osgood, *American Journal of Medical Sciences*, October, 1875.

arterial tension is only increased during the attack; the *second*, in which there is persistently high arterial tension, though still higher during the paroxysms—this class including the majority of cases."* We are cognizant that arterial spasm is brought about temporarily by the introduction into the blood of imperfectly reduced albuminoids, or their imperfect elimination; conditions brought about by constipation, imperfect intestinal digestion, hepatic inefficiency. Also by deficient elimination consequent on changes in the cutaneous circulation, or the activity of the respiratory or renal functions. Angina is, however, seldom an uncomplicated nervous disease; mostly some more or less serious organic lesion prevails, and the weakened heart cannot adequately maintain the circulation. In such cases atheroma is an almost invariable concomitant; this lesion vindicates the second division of cases of angina. An imprudence in eating, or an exposure to cold, involving a checked cutaneous elimination, may easily increase arterial tension, and precipitate an attack.†

Since cardiac disease may develop atheroma, or atheroma may induce cardiac disease,‡ we may easily have angina, a concomitant of any form of heart disease. This hypothesis is abundantly confirmed by the post-mortem table. In conclusion, a study by the sphygmograph might indicate this great predisposing cause, increased arterial tension and its outcome, atheroma. In dismissing the subject of functional disease, we pause to remark again, that any functional disorder may pave the way for organic disease, quite as surely as continued palpitation may develop hypertrophy. It is the physician's duty to render heedful attention to elaborate the causes of functional disease, and if possible, to obviate them.

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* For the observations included in quotation marks, I am indebted to Dr. Sansom's Résumé, in his Physical Diagnosis of the Heart.

† In either class psychical causes may result in increased vascular tension. "John Hunter is reported to have said, my life is in the hands of any rascal who chooses to annoy or abuse me. He afterwards died of an attack brought on by *his ungovernable* temper."—*Da Costa's Diagnosis.*

‡ *See* Observation on Aneurism, aortic disease of fatty heart.

CHAPTER VIII.

THE PULSE—THE SPHYGMOGRAPH.

A study of the pulse has received consideration in the description of the diagnosis of the various forms of heart disease. The variations in frequency in valvular disease and cardiac degeneration are chiefly in the direction of increase. A slow pulse rate has, however, been shown to accompany certain cases of cardiac degeneration (*see* Fatty Heart), but an extremely slow pulse is even more characteristic of neurosal than degenerative cardiac disease. The varying pulse rate in pericardial disease is a notable symptom of that lesion. In this section it is proposed briefly to consider the subject of arterial tension in its relation to physical diagnosis.

Increased arterial tension can be detected by auscultation and palpation, the latter method being supplemented by the use of the sphygmograph.

Auscultation discloses an increased arterial tension by the increased sharpness of the second sound (accentuation). It is naturally heard at the second right costal cartilage, if the increased tension occurs in the arterial system, and at the second left costal cartilage when increased tension occurs in the pulmonary artery. The arterial tension is increased by several conditions: (*a*) by an increased vis a tergo hypertrophy of the heart; (*b*) by renal disease, especially the *chronic* forms, with *interstitial* as well as tubular changes, also in acute nephritis; (*c*) in *atheroma of* the arterial system.

It *is the persistence* of the elevated tension which is significant of structural change.* Repletion of the pulmonary artery, with increased tension, occurs in both forms of mitral disease, often in emphysema or in pleurisy with effusion. The accentuated closure of the pulmonary segments is best heard at the second left costal cartilage.

By palpation we must note if the radial artery be normal. It may be rigid, unyielding, giving the sensation as though a piece of ipecac root could be felt under the skin. This condition of the artery is termed atheroma. A strong pulsation may be apparent on account of the rigidity of the tube, and yet if the heart's apex impulse is feeble, cardiac weakness and degeneration are indicated. When an artery is free from atheroma, the increased tension (which means the increased pressure by the blood on the inner surface of the vessels) is readily detected. The artery is less compressible; it is hard to the touch. In inflammations the pulse bears the above characters, especially in inflammations of the peritoneum and intestinal tube; but in these cases the volume is small and is termed wiry. In acute diseases of robust persons the pulse, although hard, is full. Caution must be observed not to confuse it with the *gaseous* or full pulse of an artery which has lost its tone. Such a pulse, though full, is compressible, and the percussion impulse is weak, proving that a full pulse and a strong pulse are not synonymous. The opposite, or soft, compressible pulse, implies deficient propulsive power, or loss of tone in the vessels. It is a pulse of weakness or debility. In the fevers a frequent, full, or small but soft pulse is the rule.

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* Dr. Edes has reported cases, presumably in good health, in which a high grade of arterial tension, as shown by sphygmographic tracings, is demonstrated, but it is not stated whether the high tension was a *persistent* feature.—Boston *Medical and Surgical Journal* of May 19, 1881.

Dicrotism of the Pulse.—In the healthy subject the finger applied to the radial artery is sensible of but one impulse, but it is possible to recognize one or two slight secondary elevations of the artery. These are the result of the recoil waves of blood, which, during the aortic systole, are thrown back on the semi-lunar valves, and thence rebound and travel outward to the periphery. In perfect health these waves can only be recognized by the sphygmograph, but if the tension of the artery is seriously lessened—as, for instance, in persistent high fever, 102°–104°F.—and if, further, the primary impulse produced by the systole is quick, short, but strong, the recoil waves may be felt by the finger. In individuals exhausted by chronic disease the arterial tension gives way even under moderate fever. In intermittent fevers dicrotism can often be noticed. If the pulse is very rapid, a double dicrotism may be observed.

Inequality of Pulsation.—By this it is sometimes meant that the blood waves causing the pulse do not arrive at the two wrists at precisely the same moment, but in one radial it is delayed an appreciable interval. This is a symptom of aneurism of the arch of the aorta, the orifices of the innominate or subclavian artery being altered, and the pulse delayed in the arteries of the side towards which the aneurism lies. The radial pulses may not be alike in volume, indicating a similar state, or the partial obstruction of the innominate or left subclavian, perhaps by a spicula of atheromatous deposit. It may be noticed in some cases of aneurism or aortic stenosis, that the pulse is noticeably postponed. Large aneurisms of the descending aorta, in some cases, tend to retard the pulse-wave, so that it is not perceptible in the *femorals* so soon as in the *radials.*

In inspiration the pulse-wave in all the arteries may be reduced in magnitude or suppressed, but returns to its normal volume or tension during expiration. A similar change occurs if any mechanical obstacle exist to the emptying of the left ventricle into the aorta; as, for instance, the adhesions of a fibrous pericarditis. Firm bands of connective tissue may enclose the great vessels and drag the aorta from its normal position, and attach it to the sternum. These bands are put on the stretch when the thorax is dilated on inspiration, and grasp or constrict the aorta, innominate artery, etc. The arterial pulse is rendered feeble, or even disappears, if the chest be expanded to its utmost; in expiration, on the contrary, it regains its normal calibre, and the pulse its normal volume (Guttman).

Irregularity of pulsation also includes irregularities of volume, and irregularities of rhythm. These subjects are elsewhere considered in detail.*

The sphygmograph, as applied to the study of the normal pulse, and the method of registering its results, are so fully discussed in the late works on physiology, that we are fain to present in this handbook the results. The usefulness of the instrument in the physiological laboratory is indisputable, and the facility it affords for recording cases for publication render it desirable for all to possess some familiarity with its use. In clinical medicine it has been hoped that a tracing might be made, which would enable a consultant to decide a diagnosis without a personal examination. But the great drawback to the value of the instrument is, that it is impossible to accurately gauge the pressure at which the tracings are taken. Moreover, those instruments, like Pond's, which are graduated, do not represent the exact pressure on the *artery*, since the pressure is also brought to bear on the adjacent tissues, such as the tendons of the flexor carpi radialis, the radius, skin and fascia.

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* *See* sections on Irregularities of Cardiac Rhythm and the Sphygmograph.

Tracings taken from healthy persons vary within quite wide limits as to tension. Moreover, in Pond's instrument the dicrotic wave is apt to be exaggerated by the recoil of the needle, owing to the weight of the ball attached to it. The stiffness of a spring, which receives the first impulse of the artery, also militates against the accuracy of some instruments. Discounting these disadvantages, the instrument has a great value in presenting to the eye a study of the pulse, which, united to a careful analysis of the sum of the other clinical evidence, is of much diagnostic service.

In a normal tracing* we have an unbroken line of ascent, or ventricular percussion systole, normally nearly vertical, but varying in height with the size of the vessel examined. Next we have the line of descent at an acute angle to the up-stroke. This is due to the fact that the lever falls of its own weight; but it is caught, and lifted, partly by the tide of the onward blood current, partly by the portion of it which is sent backward toward the heart by the aortic recoil (this is called the *tidal wave*). The column of blood, after impinging on the already closed aortic valves, produces a new wave,

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* For tracings see cut, at end of volume.

which takes up the descending line, forming the second curve or dicrotic wave. After which we have the period of diastole, which consists in a wave springing from the dicrotic curve (called the aortic notch, since it indicates the closure of the semi-lunar segments), and a gradually sloping line, ending at the base line of the tracing. One of these curves, however, may be wanting. Besides these recoil waves, several other more or less marked undulations present themselves in the ascending or descending lines of the tracing, caused by oscillations of the arterial wall as it expands or returns to a state of rest, after the percussion impulse.

The sphygmograph *records the frequency of the pulse.* First, estimate the time the slide takes to travel; it is usually so constructed that six inches travel in fifteen seconds; to count the pulse, measure off six inches of the tracing, and multiply by four the number of pulsations observed.

Increased arterial tension is indicated by a more or less marked deviation of the percussion stroke from the vertical; the summit is a blunt cone, instead of the normal acute angle; the first portion of the down stroke is more oblique than normal; the tidal or *blood wave* is increased; the dicrotic wave has a higher place on the tracing than is normal, or it may be but slightly marked. Low tension is indicated in the up stroke by the increased height of the percussion wave, owing to the increased calibre of the artery. Low tension is indicated in the down stroke by the more vertical descent of the first portion of the trace, and by the decided prominence of the dicrotic wave, the aortic notch being lower than in the normal tracing. When the notch extends below the level of the base line, the pulse is said to be hyper-dicrotic. Slight oscillations of the aortic walls are capable of creating undulations in the lines of ascent and descent, especially if the systole is prolonged. In aortic stenosis the percussion stroke is also more sloping, less ample, and more rounded, the increased force of the percussion stroke being neutralized by the stenosis. The less obstruction, the more vertical the line of ascent, and the greater its height, on account of the force of the impulse. If the stenosis is extreme, the recoil waves are not well marked, since the arteries are imperfectly filled, but in simple roughening or slight stenosis there is a more pronounced *tidal* wave, on account of the prolonged ventricular systole, and a dicrotic wave is present.

Aortic Regurgitation.—The up stroke is ample and vertical, owing to the powerful impulse of the hypertrophied left ventricle, and the frequent presence of atheroma (indicating enlarged and rigid arterial channels). The recoil waves are necessarily enfeebled in proportion as the aortic valves become incompetent, and the column of blood flows back into the left ventricle, since the *point d'appui* of the blood column is removed. The down stroke is prolonged and vertical, and the dicrotic wave obliterated. The presence of a dicrotic wave indicates a partial functional persistence of valve structure. The dicrotic wave, if present, appears low down in the line of descent; slight increase of pressure on the artery will again obliterate it, since the recoil in these cases is always feeble. The diastolic portion of the trace is unusually flat and prolonged, indicating arterial emptiness during ventricular diastole. An attempt has been made to differentiate, by means of the sphygmograph, *aortic regurgitation,* due to *associated degenerative arterial disease, from that of rheumatic endocarditis.* The tracing in the former case is said to indicate greater arterial tension. In our experience aortic dilatation or actual atheroma, either primary or secondary to the ventricular hypertrophy, is so constant that this distinction is of little value.

"In combined aortic stenosis and regurgitation the prolongation of the tidal wave is thought to be the measure of the stenosis" (*Mahomed*).* The presence of some dicrotic wave naturally indicates that the valves are somewhat functionally efficient.

We have described these tracings in detail in order to illustrate the following facts: that the altitude and the approach to the vertical, with an acute angle in the upstroke, is the measure of the force of the impulse, and the consequent degree of arterial fullness, with the minimum arterial tension.

The reverse is a percussion wave of diminished amplitude, deflected more or less from the vertical, with an apex appearing as a truncated cone. This tracing is an indication of the weakness of the systole, or imperfect arterial supply, or else a high degree of arterial tension.

The line of descent is vertical, marked by dicrotic curves in proportion as arterial tension is diminished; the tidal wave is increased proportionately to the amount of blood jetted into the artery, or the duration of the ventricular systole, but is reduced in proportion as atheroma or reduced arterial tension prevails. The pressure at which tracings are taken must be varied, and individual judgment must be relied on to assure the observer that the best record has been taken.

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* If the stenosis is marked, this observation cannot apply. *See* remarks on tracings of simple aortic stenosis.

If the summits of a series of percussion waves can each be touched by a parallel line, the volume of the pulses is equal.

The *regularity* of cardiac time is gauged by the regularity of the succession of tracings, both at the base, as well as their summit. In interpreting these changes of cardiac rhythm, we must remember irregularity is both functional and organic.* It is manifest, on reflection that tracings of lesions of the *mitral valve* must be very varied. If, in mitral regurgitation, the hypertrophy is compensatory, a satisfactory tracing can be secured to serve a record. In mitral obstruction the arterial tension is apt to be low, the percussion wave, however, is abridged in amplitude, on account of the imperfect blood supply to the left ventricle and absence of hypertrophy; the line of descent is very oblique in serious cases. The diastolic element is prolonged, and the evidences of *irregularity* are very frequent.† In all tracings the nearer the heart they are taken, the shorter will be the primary wave, and the recoil waves will be more pronounced and nearer the summit of the wave of descent. One must be careful to secure a tracing while respiration is being naturally carried on, since on inspiration the intra-thoracic blood pressure is reduced, and the pulse wave is reduced in magnitude.

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* For further details on this subject and sphygmography in general, *see* Dr. Mahomed's paper in the *Med. Times and Gazette*, for the year 1872. Also Dr. B. Foster's Clinical Medicine.

† Sections on Deviations of Cardiac Rhythm, and Degenerations of Cardiac *Substances and Mitral* Stenosis.

CHAPTER IX.

CARDIAC THROMBOSIS.

The subject of thrombosis and its kindred lesion, embolism, is one which merits more detailed attention than the plan of this handbook will admit. A few words must suffice.

Intra-cardiac thrombosis occurs sometimes as the direct result of endocardial inflammation, or by the partial stagnation of blood from failing cardiac propulsive power.

Various organic lesions of the heart and pericardium, and certain pulmonary lesions, mechanically cripple the heart's action. Partial stagnation of the blood current is the result of the processes, pneumonia or Bright's diseases. Certain conditions of the blood also render it liable to coagulate. *Arterial thrombosis* depends upon local disease of the arterial coats, upon pressure or obstruction, from causes external to the vessel, upon alteration of blood crasis, in which its coagulability is increased. These causes may be associated with diminished vasomotor tonus, which favors a more sluggish circulation than natural. We shall simply refer to thrombosis as it occurs in the pulmonary artery, and shall avoid the question of differential diagnosis between a homologous pathological state in this vessel, perhaps the outcome of embolism.

Cardiac Thrombosis.—The finding of clots in the heart is a very frequent incident in post-mortems. In works devoted to pathology the differences between ante- and post-mortem clots are enumerated. In making a diagnosis of the condition during life we must remember the condition is rare, save at the close of those cases of disease in which the pulmonary circulation is seriously crippled. The clot is usually on the right side of the heart. The cardiac rhythm may become very irregular, and heart sounds may be dulled or no change of rhythm may be noticed, merely a change of quality. If a murmur *preëxisted*, it may be acoustically modified or suppressed, depending on the location or distribution of the clot.

Characteristics of these Murmurs.—In murmurs due to thrombosis, the changes incident to the cardiac substance in the special valvular lesion will not be observed. Murmurs due to thrombi are developed suddenly; usually they are systolic, basic, soft and blowing, not transmitted in the route of the blood current. They are heard best over the most superficial area of the cavity in which they are produced, the right ventricle being a favorite location. These murmurs are well heard at the ensiform cartilage. The position and distribution of the clot varies, and consequently the location of maximum intensity frequently changes. This is an important symptom. (*See* Inorganic Murmurs.) The *urgency* of the general symptoms varies in proportion to the size of the clot. Dyspnœa is very prominent in the list. Next comes a rapid pulse, of course, more or less irregular in force and rhythm, amounting to 130–160; pallor, coldness, more or less tendency to syncope, and, if the clot *be very large,* all the consequences of venous congestion.

The results of thrombosis vary with the size and location of the clot. Small thrombi may undergo resolution and disintegration. We have seen cases where the diagnosis seemed to be established with a fair amount of certainty, in which recovery really took place with a *disappearance* of the physical signs. A diagnosis of this sort is naturally subject to criticism or objection.

Large clots formed suddenly destroy life in a few days, but death is partly due to the preëxisting disease, pneumonia, Bright's diseases.

Where the formation of a clot is slow, life may be prolonged for a considerable period of indefinite limits. *The diagnosis* is based on presence of the predisposing causes, upon an accession of dyspnœa and rapid pulse, without adequate explanation by analysis of the condition of preëxisting disease.*

So far as the murmur is concerned, the most valuable evidences consist in its want of transmission in the direction of valvular murmurs and the shifting position of maximum intensity.

Of the Pulmonary Artery Thrombosis.—The symptomatology includes thrombosis or plugging of the main trunk or its radicles. If the main trunk of the vessel is involved, we may have sudden extreme dyspnœa, and death. If the clot be smaller or distributed further from the heart, the symptoms of dyspnœa vary in accordance with the efficiency of the collateral circulation. It may be possible, by excluding other murmurs, on grounds already explained, or by an analysis of the predisposing causes, to establish a blowing systolic murmur, distributed in the course of the pulmonary artery, indicative of pulmonary artery thrombosis. Or, again, if infarction of the lung has occurred, we may expect to find the physical signs appropriate to condensed pulmonary tissue—signs which are appraised by their association with the predisposing causes of embolism.

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* The writer believes he has seen one or two cases of pneumonia in which a murmur developed suddenly, which he believed to be due to thrombosis. Subsequently the murmur vanished.

In conclusion, remember that embolism in any locality may be the outcome of thrombosis, and can be distributed in any part of the arterial system. In the brain the diseases of the arterial system favoring thrombosis produce increased brittleness, and also favor rupture and apoplexy. In venous trunks, in the radicles of the intestinal veins, ulcerations may originate by the agency of local inflammations (phlebitis), which may terminate in thrombosis, to be followed by embolism. From the intestine, emboli are arrested in the liver, a fruitful cause of hepatic abscess, from whence fresh emboli may be carried to the heart. From the venous trunks of the extremities emboli may be carried to the right heart and lungs directly. It is possible for the emboli to pass through the pulmonary circulation, and obstruct some artery, but arterial embolism usually has its genesis in the arterial system.

CHAPTER X.

DISEASES OF THE CARDIAC SUBSTANCE—HYPERTROPHY—DILATATION.

We shall study in this chapter the diagnosis of the lesions of the substance of the heart, not directly connected with valvular disease. In the first place, hypertrophy has been alluded to as a sequence of valvular disease. But it is also an independent process. It may affect each of the cavities of the heart; the ventricles in particular are affected, and especially the walls of the left ventricle; these may be increased from the normal half an inch to two or more inches.* In what does the lesion consist?

In simple hypertrophy the basic portion is most changed, though in aortic disease the apex is chiefly involved. In hypertrophy the special form of the heart is retained, but in proportion as dilatation is superadded the shape becomes unnatural. It is nearly always combined with *dilatation*, representing a *condition* in which the capacity of the cardiac cavities is increased disproportionately to the thickness of the walls, whether the walls be thicker than normal, natural thickness, or thinner.† It is a process often interlaced with fatty degeneration of the heart, and physical signs of both conditions are often intertwined.

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* Walls of the right ventricle are thinner than the left, the proportion between them being as 1 to 3 or 4 ("Gray's Anatomy").

† In valvular, as well as simple cardiac disease, we speak of the heart as hypertrophied (though dilatation is combined with it) so long as the hypertrophy enables the heart to maintain the circulation effectively. We say the heart is *dilated when it loses* the power to maintain the circulation adequately, although *the heart* muscle is hypertrophied.

The causes of the hypertrophic process originate in consequence of excessive ingestion of food, or use of stimulants, rowing or walking, or any excessive form of exercise.

Functional palpitation, if long continued, may induce hypertrophy, precisely as long continued exercise can develop any muscle. It is common in the gouty state, and in the forms of Bright's diseases in which interstitial inflammation predominates.* It is developed by any obstruction to the circulation in the heart as already stated. Dilatation is interlaced as an almost concomitant of the preceding, and fatty degeneration follows, or is intertwined with the process. To sum up in a few words, hypertrophy is induced by over action. Increased vasomotor tonus, from defective assimilative elimination, or from psychical influence, each severally or combined, can produce hypertrophy.

We have said that each of the cavities of the heart may be involved; the right-side enlargements, however, are secondary to the left-sided diseases, or certain pulmonary lesions, which provoke repletion of the right heart. The valves themselves undergo hypertrophy and thickening if subjected to continued overstrain; the same condition may be observed in the chorda tendineæ; the lesions are very frequently observed in the tricuspid valve; sometimes in the mitral or aortic valve structure. Hypertrophy is easily recognized. In the young, and those in whom hypertrophy has been of long standing, inspection and palpation take cognizance of a prominence of the præcordial space. A study of this region indicates the size to which the heart has attained, as compared with the normal area. The impulse of the heart is sometimes so powerful that the frame trembles, and the limbs vibrate with the shock. The apex beat of the hypertrophied left ventricle is displaced *outward*, but more *downward* than outward. The apex beat is a concentrated tap, manifested as a tolerably localized impulse, the shape of the heart is retained, and the action of the heart is regular. The pulse is indicative of arterial fullness, and a vigorous vis a tergo. We must remember that in health the right ventricle is uppermost; it is only as the left ventricle is hypertrophied, or dilated, that it becomes superficial. *Percussion* is a supplemental means of diagnosis, but familiarity will soon enable one to include these three methods, while practicing auscultation.

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* The gouty diathesis is often associated with increased arterial tension, by reason of defective elimination, etc. Sometimes even without recognizable renal changes, there is a marked tendency to hypertrophy.

In hypertrophy the outline of the left ventricle preserves its characteristic form, although enlarged. Commence percussion on the outside of the area of enlargement, and percuss inwards. As one approaches nearer to the sternum, you will note the *gradual* elevation of pitch and dullness of the quality of the resonance.*

By Auscultation.—Leaving aside questions as whether the first sound is mostly a muscular or valvular sound, remark that in hypertrophy it is duller, more prolonged and heavy. The second sound may be accentuated. Murmur may be present under the circumstances already detailed. The general symptoms described as incident to the condition are too various to reduce within

* In pericardial effusion the change of resonance is abrupt.

the limits of this handbook. We must recognize the sum of these physical signs as essential to the diagnosis of hypertrophy, since highly excited motions of the heart, in certain neurosal states, may simulate enlargement.

Dilatation of the Heart.—We must guard against confusing this condition with the former, especially when there is really associated hypertrophy. We must expect signs to vary in degree, as the walls of a dilated heart may be thicker than normal, the normal thickness, or very thin. A cardinal point is, that in dilatation the pyriform shape of the heart is lost; instead of the form of the ventricle, becomes sac-like. Inspection and palpation discover the impulse of the heart carried *downward* as in hypertrophy, but more *outward* than downward. The pulsation striking the chest is *not concentrated* and *localized,* since it no longer represents the apex beat, but more or less of the body of the ventricle impinges against the chest; the apex beat is spoken of as diffused. The impulse is weakened in proportion to the degree of dilatation, and is felt as a short, feeble slap. The impulse may also be irregular (*see* Auscultation); the outline of the ventricle can be seen to contract vermicularly. This area of impulse is the enlarged and dilated left ventricle.

Percussion.—The sac-like form, instead of the pyriform shape, is very noticeable in proportion as dilatation proceeds. The apex becomes more and more obtuse, and shades into the sac-like or globular form.

Auscultation.—The sounds of the heart become clearer and sharper if the dilatation is moderate, the thinning *of the walls seeming* to aid the transmission of the sounds to the ear, while hypertrophy seems to muffle them. When the process of dilatation is advanced, and the walls become very thin, the sounds are apt to become proportionately faint and ill-defined. Irregularity may be noted with reduplication of sounds. All irregular hearts are by no means dilated or fatty, but all cases of advanced dilatation or degeneration are apt to be irregular. The cause of the irregularity is the reduced ability of the heart to cope with the difficulties of the circulation.

The cardiac systole is unable to fill the aorta. A perfect aortic systole is negative, and the coronary arteries are, in consequence, imperfectly supplied. Moreover, the cardiac systole is incapable of squeezing its substance free from blood, and fatty degeneration is favored.

The cavities of the heart being imperfectly emptied, there is a tendency to venous congestion. There is repletion with venous blood of the right heart, the pulmonary, the portal, the chylopoetic and renal circulation. Œdema, hæmoptysis, indigestion, constipation, albuminuria result, a series of symptomatic evidences of morbid physiological action, already alluded to as sequences of mitral regurgitation. The pulse is small, and œdema manifests itself in the dependent portions of the body. Coughs, from intercurrent catarrhs, to which the system is exposed, œdema, are all aggravated if there be emphysema, a condition which in itself predisposes to right-heart dilatation.* Dilatation is the accompaniment of all valvular heart disease, sooner or later.

Be not deceived by the apparent strength of the heart's impulse, or the functional activity of all the organs while the heart is at rest, since a little exertion will lead to the lighting up of series of symptoms of dangerous or serious import.

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* *See* Cardiac Asthma.

Cardiac dyspnœa, or functional inefficiency dependent on organic heart disease, is rendered *worse* by *exertion*. If the cardiac disease is *neurosal*, *exertion* does not usually so markedly enhance the disturbance. If pulmonary symptoms are due to pulmonary *structural disease*, *exertion* (save in advanced phthisis) *does not increase* the distress proportionately to the exertion.[*] The lesson is obvious, in all cases of organic heart disease; whatever be the symptomatology, the truest conservatism is born of a treatment fundamentally including rest.

In enlargement of the right ventricle the conditions of hypertrophy and dilatation occur together. Pulmonary emphysema, or left-sided heart lesions, foster right-sided cardiac disease. The area of dullness extends beyond the right border of the sternum, over the cartilages of the third, fourth and fifth ribs, and if the right auricle is dilated, the dullness extends from the second costo-sternal junction to the third, mostly behind the sternum. Concurrent signs are the pulsation of the right ventricle in the epigastrium, the venous turgescence more pronounced than in left-sided lesion, venous pulse, and perhaps pulsation of the liver. This latter pulsation is systolic, and must be separated from cardiac pulsation in that region principally by the wide area of pulsation. The phenomenon itself is rare.[†]

Enlargements of the Left Auricle.[‡]—The normal thickness of the left auricle is from one-eighth to one-quarter of an inch. The reader must recollect that, normally, the left auricle lies *beneath* the right auricle. If enlarged, it occupies a space to the left of the sternum (outside normal area of cardiac dullness), between the second rib and the upper margin of the fourth rib vertically; horizontally, it may expand to considerable dimensions, greater in mitral stenosis than in mitral regurgitation. The ventricle may be very much depressed by the enlarged auricle, and the displaced ventricle may simulate a true hypertrophy.*

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[*] In emphysema, atrophic or hypertrophic, the amount of dyspnœa on exertion will be the best guide to the structural state of the heart.—*Fothergill.*

[†] *See* Epigastric Pulsations.

[‡] *See cases: Philadelphia Medical Times*, May 8th, 1880; also *Medical Times and Gazette, January 10th*, 1874; Flint's Disease of Heart, second edition, p. 21.

Percussion will define the tumor; *auscultation* will reveal the time of the murmur, which indicates the pathology of the lesion. Pulsation of the auricle of post-diastolic or pre-systolic rhythm is noted in some cases of mitral obstruction, and post-systolic impulse in cases of mitral regurgitation. Diastolic pulsation of the pulmonary artery is possible as an evidence of repletion, but the position for this pulsation is close to the sternum.† (*See* Pulmonary Artery.)

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* It perhaps will be enough to recall that, so far as auscultation is concerned, mitral obstruction or regurgitation are the single causes of this condition, and the greatest degree of enlargement occurs in mitral obstruction. In regard to mitral regurgitation, we hear with it a systolic murmur, but presystolic murmur may indicate both obstruction or simply roughening of the auricular aspect of the mitral valve. It is only with obstruction that hypertrophy occurs, and the same may be said of the other collateral evidences of disease accompanying presystolic murmur. The absence of the collateral symptomatology may render the diagnosis of presystolic murmur difficult.

† A convenient method of demonstrating the different centres of pulsation has been suggested. Cut two small circles of sticking plaster, about the size of a cent. Transfix the centre by a pin, so that the head is in contact with the adhesive side of the plaster; attach the plasters to the different centres of pulsation. The shafts and points project forward, and you have two levers which vibrate with the movements communicated to them by the several pulsatiens. These levers can be rendered more distinct by attaching small pieces of tissue paper as standards.

CHAPTER XI.

DEGENERATIONS OF THE HEART SUBSTANCE.

Fatty Heart.—This condition is often linked with the dilatations due to valvular disease, but may exist independently. When the blood tissue is imperfectly elaborated, or when it is inadequately depurated, the fundamental conditions of molecular life, growth, development and reproduction, are impossible. We have already shown that the condition of dilatation of the heart favors impairment of circulation, and subsequent degeneration; all dilated hearts are not fatty hearts; but all fatty hearts, from weakness of their walls, soon become dilated. We wish to speak of the etiology of fatty heart when the lesion is developed independently of valvular disease. It may occur as the sequel of atheromatous lesions in the *coronary arteries;* but atheroma of the coronaries is the expression of a more general atheromatous change, in its turn traceable to defective assimilation or elimination. This imperfect crasis of the blood finds its expression in gout, in gouty bronchitis, in eczema, in muscular rheumatism, indigestion, neuralgia; all traceable to excessive nitrogenous waste in the blood. Atheroma is developed as a sequence of this imperfect blood crasis; the point we wish to inculcate from the etiological view, is that fatty heart is but one of the local incidents of a wide spread process, which finds expression in very varied *symptoms.* The excessive ingestion of alcohol, prolonged wasting disease, notably prolonged suppurations, anæmia, all are predisposing causes. In typhoid fever we have a rapid fatty degeneration, and wasting of the cardiac muscle, a peculiar incident of the disease; besides which Dr. Fothergill has described an acute fatty degeneration of the heart, which will be subsequently given in his own language.

Fatty heart, as a symptom of general tissue degeneration, is sometimes a disease of middle life, but mostly it occurs in more advanced years, intertwined with arterial diseases, rigid, incompressible arteries, tortuous temporals, as the expression of fatty and calcareous degenerative change almost normal to old age.

After this glimpse of the associated pathological states, we must search for the tell-tale symptomatology. We have no positive physical signs of the change to be developed by a study of the heart. We may suspect it, says Dr. Da Costa, "if the signs of weak action of the heart, feeble impulse, ill-defined sounds, co-exist with oppression, with a tendency to coldness of the extremities, with a pulse permanently slow or permanently frequent, or irregular, and are met with in a person who is the subject of gout, or of wasting disease, or is very intemperate, or has arrived at a time of life at which all the organs are prone to undergo decay. Something more than a suspicion is warranted, if in addition there be proof of atheromatous change in the vessels, or fatty degeneration elsewhere, such as arcus senilis; or if it be ascertained that the patient suffer from pain across the upper part of the sternum, and from paroxysms of severe pain in the heart; that he sighs frequently; that he is easily put out of breath; that his skin has a yellow, greasy look; that he is subject to syncope or to seizures, during which his respiration seems to come to a stand still; and that he is liable to vertigo, or to be stricken down with repeated attacks having the character of apoplexy, save that they are not followed by paralysis."

This masterpiece of word painting fitly clothes the conception, and makes a tangible reality of a rather weird condition. In reference to the pulse it may be fast or slow.

Since the heart is weakened, the pulse rate may easily be slowed—just as when aconite or antimony depress the circulation. But if cardiac degeneration is extreme, the weakened muscle responds by a series of more or less abortive systoles, several failing to accomplish the results of one normal systole.

The rate of the pulse is necessarily only a corroborative sign, since a very fatty heart may indolently contract, or try to effectively accomplish the circulation by a series of abortive systoles. Exertion may precipitate an attack of palpitation, while the pulse may be very slow when at rest. In differentiating the condition by the pulse, let us say that exertion fails to excite to very rapid action a heart which is merely subject to neurosal disease, or to embarrass the organ in any way. So far as arcus senilis is an evidence of fatty heart, we must recollect that there are arcus and arcus. That arcus dependent on calcareous degeneration of age is recognized by a circle, or a semicircle, of opaque whiteness. If this be well defined, and the rest of the cornea bright and translucent, it is probably no indication of serious internal degenerative change. But the arcus associated *with a blurred*, ill-defined ring, if the ring be yellowish rather than white, the rest of the cornea slightly cloudy, you may consider that the chances of cardiac degeneration are serious; at the same time fatty heart may be present without pronounced arcus of any kind. Fatty heart is not commonly associated with increase of the area of præcordial dullness, or serious dropsy, nor local congestions, unless associated with dilatation, but the conditions are often interlaced.* In old persons, however, a moderate degree of dropsy, mostly of the lower extremities, is frequently a symptom of cardiac feebleness, and urine may show traces of albumen.

The examination of the heart, with a study of the sum of the probabilities of cardiac degeneration, is the best means of establishing a diagnosis, although the other causes of dropsy must be reviewed. Especially must we exclude dropsy due to changes in blood crasis, impairment of vaso-motor tonus, or other causes, which, to say the least, are not directly dependent on the condition of the heart, liver, or kidneys.†

Dr. Fothergill has recently described a condition of heart starvation which might represent to us acute or subacute fatty degeneration versus the more chronic condition usually understood. His language is as follows:‡ "It is a condition springing from defective assimilation: all the tissues are badly nourished, but the heart and diaphragm suffer most from the ceaselessness of their round of duty. Physical signs of feeble impulse, and weak, ill-defined sounds, are identical with fatty heart. Heart starvation is independent of widespread changes; is associated with anorexia, indigestion, deficient hepatic secretion, pale stools, deposits in urine, and the characteristic tongue. There is depression, irritability from the blood-poisoning of mal-assimilation, and the train of symptoms of deficient blood depuration we have already sketched. We may even have species of angina. The disease may pass into chronic fatty heart through development of arterial changes." This cardiac state, he believes, occurs in early and middle life, associated with overwork and deficient digestion. Dr. Fothergill thinks that prolonged debauch, during which alcohol alone is taken, may result in actual fatty decay of fibrillæ, which wear out and are not repaired, because of the absence of albuminoids in the food. A similar fatty state we are familiar with in fevers with high temperature, a lesion which is repaired when the assimilative processes are restored.*

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* Cheyne-Stokes breathing may occur in fatty heart. Dyspnœa on exertion is an important symptom, especially of right-sided fatty heart.

† *American Journal of Medical Sciences* for 1871, vol. 2. *See* paper of Dr. H. C. Wood on "Acute Dropsy, Scarlatinal and Idiopathic;" also Trans. Phila. County Medical Society for 1879, '80, page 79. Anasarca as a symptom of deficient vaso-motor tonus.

‡ *Practitioner*, April, 1881.

The essence of the disease (if post-mortem observation should establish that the Doctor's theory has a place in actual morbid anatomy) is that repair is a probable sequence, since the causes may be obviated.†

Rest is the physician's greatest resource. The ability and willingness on the part of the patient to obey the demand for rest, influences our opinion as to the duration and termination of the disease.

Cardiac Atrophy.—In phthisis, in carcinoma, in constitutional syphilis, in suppuration of bone, and also in chronic adhesions of the pericardium, cardiac atrophy has been observed. Conjecture has referred this lesion to atheromatous disease, or obstruction of the coronary arteries. It is interesting to note that atrophy, with contraction and consequent diminution of the size of the cardiac cavities, is perhaps the condition which has been called concentric hypertrophy. In adherent pericarditis we have seen a specimen in which the walls of the left ventricle equaled an inch in thickness, but the cavity of the ventricle would scarcely contain a large walnut. There are no symptoms or signs by which this condition can be positively recognized. Palpitation, or other functional disturbance, even a frequent pulse, together with the recognized existence of the predisposing conditions, are the best guides.

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* This phase of fatty degeneration of the heart is so frequent in typhoid fever, that it constitutes the cause of death in many cases of the disease in which improper exertion is allowed. During convalescence, death from cardiac syncope, or organic mischief, might follow premature exertion.

† *It is interesting* to note that the above symptoms may be purely neurosal.

Before proceeding further, perhaps a tabulated statement of the malpositions of the heart, or the causes of modifications of the cardiac sounds, may be in place.

DISPLACEMENTS OF HEART.

Upward.	Enlarged liver. Enlarged abdomen. By tumor effusion or tympany. By pericardial effusion or adhesions.
Downward.	Hypertrophy of left ventricle; of left auricle. Tumor of lung.
Laterally to left.	Dilatation—Right pleural effusion or pneumothorax; left-sided pleural adhesions. Cirrhosis of left lung. Pericardial adhesions.
Laterally to right.	Left-sided pleural effusions or pneumothorax; adhesions may form and retain it on right side. Cirrhosis of right lung. Emphysema; right ventricle most displaced.

The influence of aneurism is various. On the whole, the heart is not usually displaced unless there is valvular disease. Diagnosis of congenital *displacement is based on exclusion* and history of the case.

FEEBLE HEART SOUNDS.

First sound enfeebled.	Fatty heart dilatation. Neurosal disease. Changes in vasomotor tension.	First sound accentuated.	Mitral obstruction Aortic obstruction and regurgitation.
Second sound enfeebled.	The above conditions. Also reduced vaso-motor tonus in aorta or pulmonary artery, whether from failure of the heart or neurosal causes.	Left Ventricle.	Degenerations or dilatations of the heart.
		Aortic second sound accentuated.	Bright's diseases, chronic forms; hypertrophy of heart. Atheroma; increased vaso-motor tonus from neurosal causes.
		Second sound pulmonary accentuated.	Circulatory obstructions in the lungs. Pleurisy with effusion. Emphysema. Left-sided heart disease, especially mitral obstruction.
		Right Ventricle first sound accentuated.	The above conditions have a similar though less pronounced effect on first sound in right heart.

Pericardial effusion is a prominent cause of the enfee*blement of* the heart sounds.

CHAPTER XII.

PROCESSES AFFECTING THE PERICARDIUM.

In lesions affecting the endocardium the predisposing causes of inflammatory processes are important aids to diagnosis. In pericardial disease a true estimate of its etiological genesis is so fundamental as to be an integral part of each diagnosis.

In the outset, then, we may say that pericarditis is almost never idiopathic; on the contrary, it is developed in direct association with rheumatism, with Bright's diseases, or as an inflammation propagated from an allied process in contagious tissues. It is also a complication of pyæmia. Practically, pericarditis is mostly a local development of the first two processes. The sources of inflammation likely to occasion pericarditis by contiguity are, first, the inflammations of the pleura, especially of chronic type. It may also be the result of inflammation in the mediastinum, or abscesses beneath the abdominal diaphragmatic surface, developed by a perihepatitis. Or, pericarditis may ensue, in the course of a chronic peritonitis.

In a variously estimated proportion of cases both peri- and endocarditis exist.

Like other serous membranes, the expression of the inflammatory process occurs in two forms, the acute, with effusion, either serous or fibrinous, and the chronic, which mostly results in adhesions. In the latter form we shall have to study the effect of pericardial disease upon the cavities of the heart, since these effects are interlaced with the physical diagnosis. In formulating a diagnosis of *acute pericarditis,* consider in the first place the etiology; afterwards examine each case by one or all the methods which we shall detail. A diagnosis can often be made by a skilled investigator at a glance, or by the use of a single method of physical examination. In this respect we observe that not every case will demand a diagnosis by exclusion. There is no objection to these rapid diagnoses, save that in such cases the mind must be kept in attitude to appreciate a new grouping of the symptomatology, and in readiness to adopt a diagnosis, based on a rigid analysis.

Acute Pericarditis with Effusion.—One of the earliest evidences of pericardial effusion is by means of *percussion*. The first expression consists in the altered *contour* of the area of cardiac dullness. In health it is pyriform or pyramidal, with the apex *downward,* centering at the *apex-beat.* In pericardial effusion the *apex is upward,* the base of the triangle *downward.* The area of flatness commences below, at the sixth rib, extending upward a distance varying with the amount of effusion, perhaps as high as the first or second rib.

Following a principle involved in the examination of pleural effusions, the *transition* from the outline of dullness to the outline of resonance is *abrupt.* The edge of the lung which normally overlaps the cardiac area is pushed aside, and the area of impaired resonance is abrogated. The percussion note is really flat, not dull, *and the resistance to the fingers in percussion is most perceptible.*

There are certain facts which separate the flatness of pericardial effusion from the dullness or flatness of an enlarged heart. The first of these relates to the change of *contour* already noted. We find the apex of the triangle above, but it is a *truncated cone*, instead of terminating as the normal cone; *below* there is a breadth of dullness interposed beneath the apex beat and the area of gastric resonance.*

The amount of a pericardial effusion varies, with comparatively short intervals of time, from day to day—almost from a morning to an evening advances or recessions of dullness can be defined. A very important aid is the change of the area of flatness when the position of the patient is changed from side to side, while at the same time auscultation or palpation will show an *unchanged apex beat.*† Further examination in the recumbent position will give a clearer percussion resonance, and the line of transition will be less abrupt as the lungs return to the position from which they were displaced by the fluid.

So far as percussion is concerned, this examination merely assures us that there is fluid in the pericardial sac; this may occur equally as a passive dropsy of the pericardium, or as a pericarditis. Dropsy occurs in this situation together with other general manifestations of effusions elsewhere. For pericarditis we have, as already observed, the etiology. You will note that in what has been said an enlarged heart might be confounded with a pericardial effusion; attention to the etiology, the evidences of physical diagnosis, as herein detailed, will usually protect one from error.

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* Respiratory percussion, on full inspiration, may be made available, if the patient is not too short of breath, to define more sharply the line of transition from resonance to flatness. In cases of simple enlargement of the heart, the area of cardiac dullness is diminished by use of this method.

† Examine apex beat by auscultation or palpation.

Palpation.—In pericardial effusion, both passive or inflammatory, the study of the apex beat shows its displacement, or it is effaced. It is pushed upward by the effusion as the level of fluid ascends. When felt in the new position, the impulse is feeble, evidently distant and transmitted, and in some cases it is imperceptible. The position of the patient influences our study materially.

In the first place, if possible, incline the patient's person forward. This (if the manœuvre be admissible by the patient's condition) will render the apex beat more plain. Again, as the fluid increases or diminishes, the apex beat may become less distinct or more apparent. (*See* Percussion.) The position of the apex beat will often settle the question between pericardial effusion and enlarged heart. *Palpation* is also helpful in determining an evidence of pericarditis when *differentiating* the condition from *passive effusion.* This is accomplished by noting the presence of friction fremitus in the former case. This fremitus is felt in cases of pericarditis with moderate effusion, and a true rubbing fremitus can be distinguished accompanying either the systole or diastole, or both. In the latter case the to-and-fro character is marked.*

It is important to recognize the rhythm of such frictions as distinctly cardiac. For this purpose the patient should be commanded to hold the breath, so that one may be certain that the sensation is not communicated by the lungs or pleura. The fremitus is felt if the ex-

* *See* Thrill, Auscultation.

amination is made in the early stages of pericarditis, or else in chronic cases. The fluid effused must be rich in fibrine; the flakes must be spread over the surface of the pericardium, especially near the base, and, as polished by the cardiac action, they give at an autopsy the appearance of down or fur all over the heart. This has been compared by some to a cat's tongue. In order that these frictions may be recognized, we repeat that the amount of fluid must not be so great as to separate the heart from the chest wall, and pulsations must be sufficiently strong to cause a definite rub. Both pericardial and pleural friction fremitus can be recognized by auscultation as well as palpation, but thrill (as the friction is sometimes termed) can be remarked in some instances when friction is inaudible.

The Pulse of Pericardial Disease.—In examining the pulse certain facts must be borne in mind. On the one hand, an embarrassed or degenerated heart contracts with weak, feeble systole, and may result in a very slow pulse. On the other hand, the pulse may be very rapid or feeble. This means that the heart is either very much embarrassed or degenerated, and that several systoles are required to raise the arterial pressure to a degree of measurable physiological tone. We cannot, however, appraise the extent of the embarrassment or degeneration by the character of the pulse. Extreme degeneration may yield a very slow pulse, or at other times a rapid pulse. Slight degrees of degeneration will often slow the pulse rate to a marked degree. In pericarditis the embarrassment to cardiac rhythm may be considerable, insignificant or varying. This explains the observation of Dr. Walsh's, viz., sudden *variation* of the pulse rate. In pericarditis a very slight movement of the body increases the pulse from 60 to 90 or 140. But since neurosal or emotional conditions may cause variations of the pulse rate, the value of this symptom is only in connection with other signs.

Auscultation in acute pericarditis reveals two separate series of signs in natural sequence to the condition of the pericardial surface. Sedulously practice auscultation in all cases in which etiological conditions suggest the possibility of pericarditis. If there be a fibrinous inflammation, a rubbing or creaking sound may be audible, accompanying both movements of the heart. Sometimes the murmur is a soft, blowing sound, resembling the attrition of two surfaces of cloth, or the murmur may resemble so closely a valvular murmur that acoustically they are identical. The friction may occur rhythmically with one or other sound of the heart, but much more commonly it is a to and-fro sound.*

Among the peculiarities of the pericardial friction sound are, that it is very transient. Walsh says that it may appear and disappear in six hours. It may disappear on account of the increase in effusion, to reappear and finally vanish gradually. Most commonly, however, friction sound is audible for several days. It is a very common incident for a friction sound to occur in a case in which an autopsy will reveal scarcely any lesion (pericarditis sicca). The characteristics of a pericardial friction sound are chiefly: 1st. Its change of location of maximum intensity, since when one mass of lymph may be polished smooth other roughness may form. 2d. It is limited to the cardiac area, and is not propagated in the line of the transmission of valvular murmurs. 3d. It is synchronous with the movements, rather than the sounds of the heart. Commonly, it is heard over the base of the heart, or near the orifice of the vessels. 4th. It is a superficial sound; the valvular sounds are heard beneath.* 5th. The change of posture may induce an increase of murmur, rendering a murmur which is *faint* in the vertical position quite distinct in the recumbent. 6th. The alterations common to the walls of the heart in endocardial disease are absent. This point will not be of great value, since endocarditis is often associated.

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* *See* Palpation.

Differential Diagnosis.—The first source of error consists in a confusion of pericardial frictions with those in the pleuro-pericardial.

In this case the balance of etiological probabilities is of great importance; but this, while serving us when rheumatism is the antecedent, fails us when Bright's diseases exist, since in these diseases there is a predisposition to inflammations of all serous membrane. Is it of respiratory rhythm, or is it more pronounced at end of full inspiration? Is it a murmur of variable intensity? A pericardial friction varies less than a pleuro-pericardial.†

However much the pericardial murmurs resemble the endocardial, the latter can be recognized by the depth of the plane at which they are developed, by their transmission in the route of the blood current, and by their being in association with definite lesions of the heart muscles.*

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* The importance of recognizing the plane on which abnormal sounds are developed was dwelt on when considering the question of pleurisy.

† Walsh refers to a friction produced by the pulsation of the aorta, which is diastole in rhythm, or an exo-cardial friction between the aorta and the pleura. The diastolic rhythm and increase during full inspiration may help to establish their identity.

Now, as for the second group of auscultatory signs, viz., pericarditis with effusion, the sounds of the heart, especially the first, are very faint and muffled, sometimes almost inaudible. If the effusion is not too large the sounds are clear over the apex beat, which has been elsewhere shown displaced upward, and the sounds grow louder as the stethoscope is carried upward.

We notice, in some cases, considerable dyspnœa; in others none. Certain postures may give relief; for instance, if the recumbent posture on the left side is selected, the liver and heart both tend to exercise pressure on the pericardium, so that the posture on the right side is apt to be preferred. As for pain, there is no more delusive symptom, since an entire attack of pericarditis may transpire without a lament from the patient.

Inspection includes a possible bulging of the præcordia and filling out of the intercostal spaces, together with the already often repeated displacement of the apex beat upward, and outward, perhaps, into the fourth interspace. The impulse may be feeble, weak or fluttering.

Differential Diagnosis.—The only source of error seems to be the possibility of mistaking a dilated, fatty heart, with feeble heart sounds, for the enlargement of pericardial effusion. The simple mention of this source of error should be sufficient to prevent a mistake which may be avoided by attention to the general methods of investigation.

---

* The respective dates of the commencement of the endo- or pericardial disease *must be determined* chiefly by the history.

In differentiating pleurisy with effusion, note that the physical signs of the disease in the pulmonary apparatus can be noted in the back. In anterior sacculated pleurisy the position of the collection will best serve us when contrasting the symptoms.

The Terminations of Pericarditis.—The issue of a case of pericarditis may be summed up as follows: Death may occur, on account of the amount of pericardial effusion, especially if pericarditis complicates the condition of a heart already weakened by endocardial disease or fatty degeneration.

In Bright's diseases the prognosis is also unfavorable. *General Symptoms.*—Wild delirium may be an accompaniment of pericarditis, and the præcordia should be examined whenever the symptom complicates rheumatism or Bright's disease.* Associated gastric irritability is also, sometimes, a symptom liable to divert attention from its proper localization. At the risk of recapitulation, we would again call attention to the frequent transitory character of the attack, and especially of that important symptom, friction sounds. These being produced sometimes by mere prominences of vascular tufts, with but insignificant exudation, may be relieved by treatment, and all signs vanish in a few hours.

Chronic Pericarditis.—The limits of these pages only permit a cursory mention of this interesting phase of pericardial disease. When a patient comes into one's presence with chronic heart disease, one must always ascertain if symptoms be really closely connected with the lesions detected by physical diagnosis.

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* When delirium, with more or less stupor, occurs in acute rheumatism, the symptoms may be due to the elevated temperature, the so-called cerebral rheumatism. The temperature in these cases is always very high. In cases reported under this reference, temperature in a case of rheumatism suddenly rose to 108¾° F. Phila. *Medical Times*, May 30, 1874; Clinical Lecture on a case of Cerebral Rheumatism, so called; use of cold baths. By H. C. Wood, M.D.

In all our studies of the heart a strict censorship must be maintained, to obviate a disposition, on the one hand, to disregard the complaints of a patient, through an enthusiasm over the physical signs of disease, or else, in chronic cardiac disease, to attribute all the complaints more or less directly to the heart. In no condition of chronic heart disease is the above caution more applicable than in chronic pericarditis, since it may produce many serious results, or the lesion may be devoid of inconvenience. The lesions of chronic pericarditis are very similar to those of the pleural serous membrane. 1st. The effusion may remain without tendency to increase or diminution. 2d. In adhesion or agglutination of the pericardial surfaces. This latter lesion induces, as sequelæ, either cardiac hypertrophy and dilatation, or cardiac atrophy. The first class of lesions require no elaboration, since the signs of effusion are always the same. The second condition is one of the hard problems of physical diagnosis. Again, at the outset, we require an unequivocal history.

We believe that dyspnœa, of the form peculiar to the heart (viz.: increased by exertion), is a sign of chronic pericarditis with adhesions, provided that careful study excludes any other form of cardiac or pulmonary disease, or any neural disturbance of the heart. In hypertrophy connected with pericardial adhesion the heart's apex may pulsate as high as natural, in spite of the enlargement, or the heart may be drawn *upward* and *outward*, to the *left*, as found by percussion. This is not the history of *hypertrophy* and dilatation without adhesion.*

---

* *See* Hypertrophy and Dilatation.

If the pericardial surfaces are extensively united, the præcordiæ may be depressed and the interspaces drawn inward during the systole. The heart's apex may be drawn upward and outward; even the lower portion of the sternum may be contracted.*

The limits of cardiac dullness may be enlarged and unchangeable in varying postures of the body, or in full inspiration and expiration. There may be no change in the rhythm of the heart, or there may be a very *variously expressed alteration* of *rhythm,* including irregularity. A possible undulating movement over the præcordia is unreliable, because it is often seen in cases of enlarged heart without adhesions. We may fairly say that the diagnosis can only be effected by taking into the fullest consideration the predisposing etiological causes and the sum of the physical signs just laid down, together with a general diagnosis by exclusion.

The same may be said of the diagnosis of friction sounds between the pericardium and the pleura, called pleuro-pericardial frictions. If the results of other physical signs favor pleurisy, the friction may have a pleural region. There is one sign which may be conclusive, viz., the holding of the breath, which will arrest the friction sound if it be of respiratory rhythm. Yet even this sign may fail, since the heart may disturb the surface of the pleura and develop a friction.

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* Walsh thinks that the systolic dimpling of the interspaces only happens if there be, in addition to pericardial adhesions, pleuritic adhesions in front of the organ, or if the agglutination of the pericardium be combined with cardiac hypertrophy. We would refer, in this connection, to what was said, under the head of Emphysema, in reference to the loss of resiliency of the distended chest, and the liability to depression of the interspaces during inspiration. We have also seen the dimpling of the interspaces and wave-like vermicular tremor of præcordial spaces, in pericardial adhesions, without pleuritic adhesions.

INDEX.

PAGE

Aneurism—diagnosis.............. 149
" murmur in............ 196
" without murmur....... 197
" abdominal............. 198
" varieties........... 192–194
" position in......... 199–200
" terminations of........ 201
" position of apex beat... 242
Aortic disease—causes of...160, 168–184
" " diagnosis.......... 184
" " general symptoms.. 189
" " quality of murmur 168
Arterial disease 160
" tension (see also Tonus).... 182
Accentuation of aortic second sound 216, 217, 242
" " pulmonary artery, second sound... 181
Atheroma—diagnosis of.. 184, 192, 189
" in angina pectoris...... 214
Angina pectoris.................. 212
Adhesions of pericardium......... 254
Asthma, cardiac................... 181
Asthma, renal................ 112, 114
Apex murmurs..................... 170
Arterial " 205
" " in incipient phthisis 205
Auricular enlargement.........172–175
Auscultation of heart............. 163
" " lungs, method of, 32, foot note 59
Abscess of lung (see Cavities)....
Amphoric voice and breathing 87,133,147

Bronchitis, acute and chronic...108–109
" capillary.............. 110
" associated with catarrhal pneumonia........... 47
" associated with cardiac disease.............. 181
Bell-sound in pneumothorax....... 149
Bronchophony..............34, 46, 134
Bright's diseases—influence in pericarditis....................... 252
" " influence in heart disease..161, 163, 252
Bruit............................ 197

Cavities, in the lungs............. 79
" *due to abscess*............ 82
" *location of*..............80–81

PAGE

Cavities, in bronchial tubes........ 82
" in phthisis 83
" percussion of.....83, 84, 85, 86
" auscultation of........... 87
" mistaken for pneumothorax 151
Cracked-pot resonance............. 84
Cancer of the lungs............... 152
Cheyne-Stokes respiration......114, 213
Corrigan pulse................187, 195
Carotid pulsation.................. 190

Dilatation of heart with mitral disease, regurgitation..... 172
" of heart with aortic disease...............188, 189
" (see Fatty Heart).
" with mitral obstruction or roughening 179
" with tricuspid disease 175, 180
" of the heart............. 232
Dropsy with mitral disease........ 172
" " cardiac disease....... 162
Diastolic murmur.................. 186
Displacements of the heart, especially of apex beat....127, 241, 242
Dyspnœa, cardiac...........211, 234, 253
Death in mitral disease.........173–188
" " aortic " 188
Disappearance of presystolic murmur......... 183
" " tricuspid murmur 174
" " mitral murmur . 171

Expiration in emphysema......... 92
" importance of high pitch 70, 92, 110
Emphysema, vesicular............ 64
" with phthisis, vesicular 68
" substantive, general.... 89
" diagnosis.............91–96
" atrophic............. 97
" (see Respiratory Percussion).
Empyema 144
" surface thermometry....146
" diagnosis 147
" pulsating............... 141
Epigastric pulsation................ 95
Endocarditis 160, 206
Etiology of cardiac disease.... 161, 244
Effects of valvular disease........ 171
Embolism (see Thrombosis).

PAGE
Fremitus, vocal normal............. 34
" " increased.. 46, 49, 60, 70
" " absent.............. 130
" " diminished.. 70, 93, 143
" " friction (see Thrill).
Functional cardiac disease......... 207
Feeble breathing, significant..... 68, 77

Gangrene.......................... 80

Heart, hypertrophy..........160, 229
" " in mitral disease 170
" " in tricuspid " 180
" " in the aorta.... 188
" " with dilatation.. 171
" " " mitral obstruction..... 179
" " of auricle....... 235
" " of right ventricle 234
" atrophy.................. 241
" displacements of.......241–242
" " of apex in chronic pleurisy and fibroid phthisis............ 127
" angina pectoris............ 212
" pain (see Angina),
" irregular, intermittent..207–208
" thrombosis................ 225
" dilatation (see Dilatation).
" fatty degeneration......... 236
" table of causes of feeble sounds.................. 242
" starvation................ 239
Hydrothorax...................125–139
Hæmothorax...................... 147
Hydro-pneumothorax 148

Interrupted or cog-wheeled respiration........................... 59
Inspection, normal................ 37
" in early phthisis...... 60
" in advancing " 71
" in emphysema........ 147
" see pleurisy 129
" see emphysema....... 94
Inorganic murmurs.......203, 225–227
Intermittence of cardiac rhythm.. 207
Irregularity " " "...... 208
Intercostal tissue, movements of 94, 128–129 over 94, 128–129
" præcordia 254
" neuralgia 77, 138

Mensuration, rules for performing 35
" in phthisis 75
Movements of intercostal tissues 94, 128–129
Metallic tinkle in pneumothorax.. 150
Murmurs, definition 163
" laws governing........ 169
" mitral regurgitation 168–169
" " disease exceptions 170

PAGE
Murmurs, mitral disease complications. 172
" " cause of death 173, 180
" " prognosis 173
Mitral disease, obstruction........ 177
" " double murmur.... 183
" " presystolic murmur 178
" " secondary to aortic 189
" " causes of and character of murmur 168
Murmur, tricuspid............. 173, 177
" aortic disease, single and double................ 185
Murmurs due to aneurism.... 193, 196
" relation of to sounds..... 190
" due to aortic valvular disease contrasted with aneurism............. 193
Murmur called bruit.............. 197
" functional....... 203, 225, 227
" pericardial........... 247, 249
" influence of posture on 171, 174, 183

Neuralgia, intercostal......... 77, 138
Neurosal heart disease............ 207

Obstructive valvular lesions, aortic 185
" " " mitral 167, 177

Pigeon breast...................... 40
Pneumonia, croupous, acute....... 43
" catarrhal, " 47
" râles in...... 63, 101, 108
" terminations.......... 49
" hypostatic............ 69
Pneumothorax.................... 147
" percussion, metal sound........... 149
Pleurisy, acute................... 123
" diagnosis............... 126
" " differential 137, 138
" chronic.............. 140, 144
" accentuation of pulmonary artery, second sound............ 180, 242
Phthisis, broncho-pneumonia... 42, 55
" fibroid............. 42, 55, 73
" incipient............. 58, 106
" chronic............ 52, 61, 66
" contour of chest.... 37, 71, 74
" venous and arterial murmurs.................. 205
" advancing............ 61, 66
Pleurodynia (see also neuralgia intercostal)........................ 138
Pectoriloquy.................. 87, 88
Percussion, method 17, 19, 24, 59, 64, 86
Pericarditis, acute diagnosis....... 245
" thrill............ 247–249

PAGE

Pericarditis, pulse 248
" friction (See Thrill). —
" position apex beat 246–247
" differential diagnosis 250–251
" with effusion........ 251
" posture in, and pain in 251
" terminations 252
" etiology, value 244
Pulsation, evidence of aneurism 195–198
" " " cardiac hypertrophy (See Heart).
Pulsation, evidence of cardiac dilatation (See Dilatation).
Pulsation, evidence of arteries 202–190
Pulse, slow...................... 216
" unequal.................. 218
" irregular, 218. See also intermittence and irregularity of rhythm.
Pulse, sphygmograph 219
" mitral disease 183
" aortic " 187–195
" aneurism.................. 196
" in veins.................... 176
Palpitation.................. 190, 210
Pulmonary artery sound, increased 180
" " aneurism....... 201
Presystolic murmur.............. 178
" " exceptions 183
Pain. See pleurodynia, intercostal, neuralgia, angina.

Resonance, normal.............. 1–23
" distribution of normal 24
" vocal 33–37
" " increased 46, 49, 60, 70
" " absent 133
" " diminished 70, 93, 143
" osteal 64
Rhythm, respiratory....... 67, 76, 114
" feeble movements..... 77, 68
Respiratory rhythm, normal.... 27–30
" murmur, distribution of normal 31
Râles—classification 99
" sibilant and sonorous...... 101
" amphoric 101, 150–151
" size of 100
" crepitant, dry, and moist 101–102, 107
" friction...... 102, 133, 249, 247

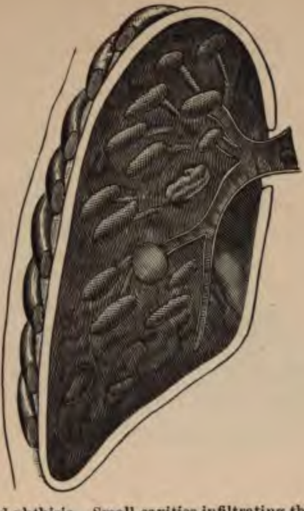
PAGE

Râles, diagnosis between friction and intrapulmonary 103–104
" due to softening, to bronchitis 105–108
" in incipient phthisis 106
" mucous 100, 107
" in capillary bronchitis 110
" in œdema 111
" in asthma................ 112
" in croupous pneumonia.... 63, 101, 108
" in catarrhal " 48, 63, 108
" in moist crackling, foot note page.................. 60
Respiration, Cheyne-Stokes........ 114
Respiratory percussion............ 156
Rheumatism in etiology of cardiac disease.....................160, 244
Renal disease in etiology of cardiac disease 161
Reduplication of sounds 181, 209
Regurgitant valvular lesions (see mitral tricuspid aortic disease). 167

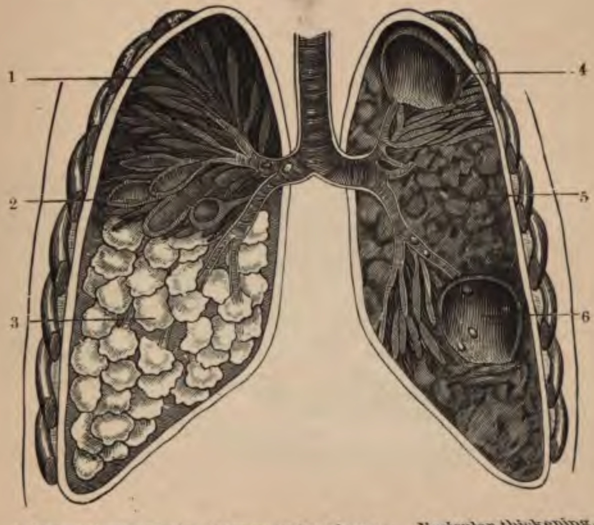
Sphygmograph 219
" its use............ 221
Stethoscope...................... 32
Syphilis, in the lungs............ 56
" relation to aueurism 193
Sounds, definition............ 163–165
" relation to murmurs...... 190
" of heart accentuated enfeebled. Classification.. 181, 216, 217 242,
Systolic murmurs,....169, 173, 185,193
" " aortic stenosis (see Obstruction).

Thrill....................... 190, 195
" pericardial.................. 247
Thrombosis.................. 225–227
Tricuspid lesion.............. 173–176
" " secondary 173
Tuberculosis, acute miliary....... 116
Tumors of mediastium............ 152
Tension or tonus in arteries (see Pulse, Sphygmograph, accentuation of aortic and pulmonary artery sounds). 217

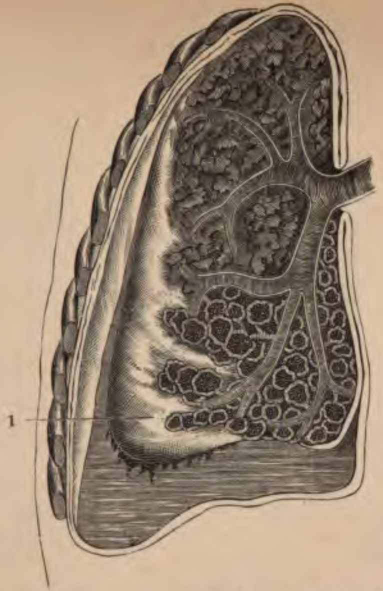
Venous pulse..................... 175
" murmurs 204
Valvular lesions, summary......... 167



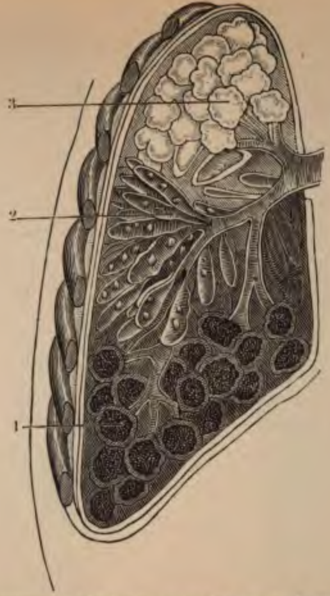
Diffused phthisis. Small cavities infiltrating the lung.



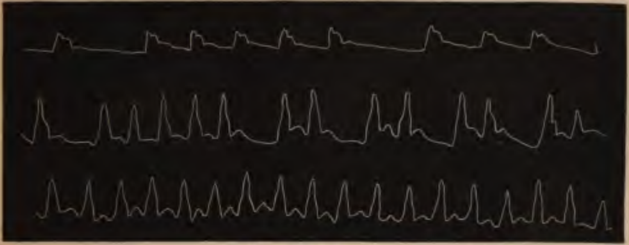
1 Catarrhal pneumonia—inter-vesicular changes. Vesicular thickening, and *collapse.* 2 Vesicular Emphysema, in patches overlying areas of consolidation, or intermixed with such areas. 3 Vicariously acting lung. 4 Formation of cavity. 5 Consolidation. 6 Large cavity—amphoric râles or jingles; also, *bronchial tubes.* See RÂles.



Pleurisy, with effusion; condition of pleura; also condensed lung above; patulous bronchial tubes. 1 Croupous or Vesicular pneumonia. The drawing, therefore, represents the process known as pleuro-pneumonia

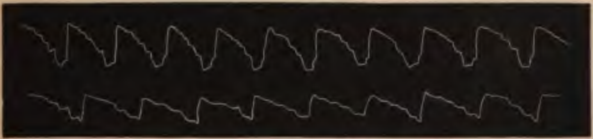


1 Croupous or Vesicular pneumonia; stage of consolidation; 2d stage, so called. 2 First stage; crepitant râle. 3 Supplementally distended vesicles.



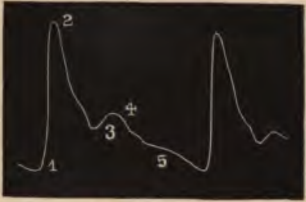
(AFTER SANSOM.)

Irregularities in time pulse tracings of low tension.



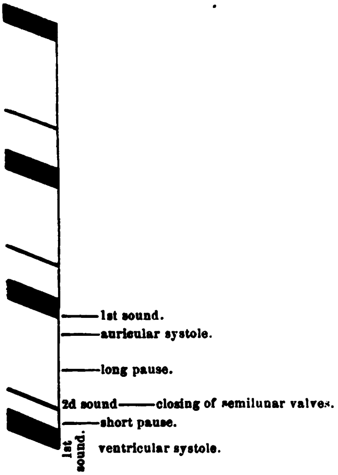
(AFTER SANSOM.)

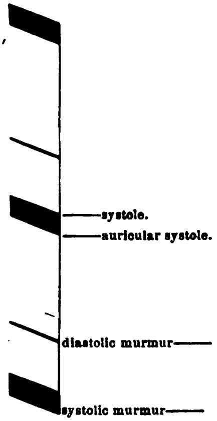
Pulse tracings of high tension.

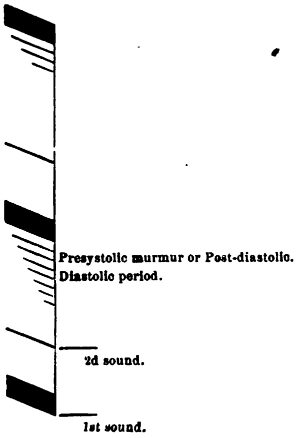


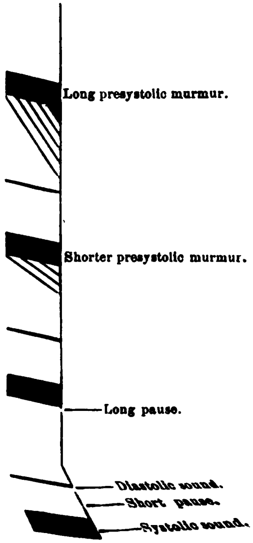
(AFTER FOSTER.)

1–2 Percussion up stroke.
1–2–3 " wave.
3–4–5 Tidal wave.
4–5 Dicrotic wave.
5 To base line, diastolic period.


1st sound.
auricular systole.
long pause.
2d sound
closing of semilunar valves.
short pause.
1st sound.
ventricular systole.


systole.
auricular systole.
diastolic murmur
systolic murmur


Presystolic murmur or Post-diastolic.
Diastolic period.
2d sound.
1st sound.


Long presystolic murmur.
Shorter presystolic murmur.
Long pause.
Diastolic sound.
Short pause.
Systolic sound.

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